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VETERINARY MEDICINE

October, 1929

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Cattle Practice Issue

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VOL. XXIV

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D. M. Campbell, Editor A. T. Kinsley, Associate Editor

A Cattle Practice Issue

The enthusiastic welcome accorded the three special issues of VETERINARY MEDICINE that have been published during past two years, each on a single branch of veterinary practice, make certain that an issue devoted exclusively to cattle practice, probably at this time the most important branch of veterinary practice, will serve a useful purpose.

The subject of cattle practice includes so many different matters that it has been impossible to include it all in the space available in a single issue of VETERINARY MEDICINE, even in the very concise discussions in which the various subjects of necessity have been couched. The whole subject of abortion and sterility, diagnosis of pregnancy, most of the discussions on surgery and some others although a part of this discussion of cattle practice will be published in the next issue.

The distinguished authorities on veterinary medicine whose discussions make up this issue and those that are to follow deserve even better from our readers than may appear at first. It is a task of no small difficulty to condense a treatise to the degree that has been maintained in these discussions and yet omit no essential part. The injunction to be brief, to be concise and yet comprehensive in scope has lain heavily upon most of these writers; increasing the task almost in the degree that the output has been decreased.

Of course the credit for whatever merit readers may find in these discussions is due entirely to those who supplied them. It is the present writer's conviction that there has not heretofore appeared in any issue of any periodical an equal number of equally high class discussions on a phase of veterinary practice that concerns a larger number of veterinarians to a larger degree than any other.

With the discussions that appear in this issue and those on the subject of cattle practice that are to follow next month more than fifty of the most important conditions that confront veterinarians who do cattle practice are discussed in a thoroughly up-to-date and authoritative manner. The discussions are not and are not intended to be exhaustive. Their purpose is to make readily available to the practitioner the essential facts on the subject with sufficient completeness to enable him to put into practice the best known methods of diagnosis, prevention and treatment for the conditions discussed.

Physical Examination of Dairy Cows

By S. L. STEWART, Olathe, Kansas

PHYSICAL examination of dairy cows is second in importance only to tuberculin testing, and since the percentage of cows infected with tuberculosis is low in many sections (in this county is only a fraction of 1%) it is a question whether the physical examination does not take first place in importance.

It is necessary only to name over conditions, "diseases" found and eliminated by the physical examination which are undesirable and in some cases render the milk positively dangerous to human health, especially to infants and children, to emphasize its importance.

Conditions usually found on the first physical examination of a dairy herd are: mammary abscesses, both superficial and deep; acute streptococcus or staphylococcus infection of either the parenchymatous or interstitial tissue of the udder; gargety udders, especially pus producers; purulent metritis; punctured sloughing wounds, especially between the two front quarters; sloughing wire cut teats; cow pox; actinomycosis; leukemia and anemia. Other ailments may be found, such as pneumonia, pleurisy, indigestion, sore feet, etc.

High prices are paid for sanitation and sanitary surroundings in our hospitals for the sick, and for infants as well. Sanitary food, as far as possible, is used; then why not have milk for hospitals at least that comes from cows that have passed a rigid physical examination to insure as clean milk as possible for the sick people and babies. They are entitled to it.

Method of Making Examination

In preparing to make a physical examination of dairy cows, the veterinarian should clothe himself in a large suit of unionalls, a pair of rubbers for the shoes and have ready blank records to be filled out.

When the cows have all been milked, the examination should begin.

Approach the cow on the right side, lay the left hand on the back, then while the cow is getting used to the stranger, an observation is made of the side of the head, neck and shoulder. Then the right hand is placed on the withers and with a firm pressure brought down over the region of the prescapular lymph glands. Then, facing backward, the right hand is placed on her back, the left hand is then passed down over the prefemoral lymph glands, and then placed on the udder, over the teats, lifting up and at the same time feeling for deep abscesses. Then the fingers are drawn from behind forward in the groove between the two halves, to ascertain if punctured wounds are present, which always have a very offensive odor in that region. Next, the left hand is placed over the back quarter of the udder with a firm pressure and slowly moved upward to the perineal region to palpitate the two mammary lymph glands.

This completes the examination on the right side of the cow. The same examination is made in the same way on the left side. The same examination is made on every cow that is giving milk and on those that will freshen soon. Then a final observation is made of the tail and buttocks of all the cows for sticky pus on the hair, which is an indication of purulent metritis. The examination takes only about one minute to a cow. Then the herd record must be made.

The record should show the ear tag number, breed, color, weight, age, date of last calving or next calving time of each cow, and last, but most important, if the udder is normal or diseased. Then remarks, if necessary.

All cows that are diseased should be removed from the herd. There are only two methods of disposing of cows that have been rejected. All those that can not be cured should be sent to slaughter at once. Isolate the others for treatment.

The qualifications of the examiner are of much importance. Only veterinarians can intelligently make such important examinations, diagnosis and prognosis of the various diseases and conditions found and the tissues involved. In fact, a diagnosis of the fitness or unfitness of a cow for the production of milk for human consumption is far more important than diagnosis and treatment of disease, as then only the health and life of the animal is at stake, whereas the health of people, especially infants and children, is at stake where the physical examination is not made by properly trained veterinarians, or where no physical examination at all is made. Therefore such examinations should never be intrusted to any one but a veterinarian.

The Kansas City Health Board is in possession of data which proves beyond a doubt that the physical examination of dairy cows is a positive asset in the production of good milk and consequently to the health of children. It is also a decided asset to the dairy industry. The Kansas City Health Department regards veterinary inspection (physical examination) as having been directly responsible for lowering the bacterial count of milk nearly 50% since the physical examination of dairy cows was inaugurated in 1923.

If the figures of the Health Board are correct, the physical examination of dairy cows by veterinarians and the elimination of the cows that were not physically fit for production of market milk have done more good than all the other requirements combined to reduce the number of bacteria in milk.

Profits in Physical Examination

Taking up the dairyman's side of the proposition first, these facts are found, that within a few months his profits have

PIETERTJE

This cow, the property of the University of California, produced 28,235 pounds of milk testing 1000.4 pounds of butter fat in 1923. Note the near approach to the ideal conformation for a dairy cow.

been increased by the physical examination. He profits financially in three ways:

1. By disposing of all cows with spoiled udders he checks, to a very large degree, streptococcic and staphylococcic infection of the udders of other cows.

2. He profits when he disposes of the condemned cows and replaces them with cows with healthy udders, which are high milk producers. At the present

An extreme case of abscesses in the udder. Milk from such cows is a frequent cause of a high bacterial count in the mixed milk of a whole herd.

price of feed, it costs about 45c a day to feed a dairy cow. If she produces six gallons of milk a day and the milk is sold in cans, it is worth $1.20, or 75c profit. If she averages that amount for only 240 days, she has made for the dairyman a gross profit of $180.00,—sale of the calf not included. In the same cow, with an acute streptococcic invasion of the parenchymatous tissue and a loss of only one-quarter, at the time of calving the milk production will be decreased usually to at least one-half, or 3 gallons per day, for the same period of time, "but she usually will not milk that long." Her gross profit will be $28.00 for the 240 days, as against $180.00 for the normal and healthy cow. Frequently the diseased cow will not pay her feed bill for a year, besides the danger of spreading the infection. If her milk is sold bottled as A grade raw milk it will be worth at least $675.00, for the cow with the healthy udder, whereas it is very likely worth less than the cost of her feed if the udder be diseased. Taking

Bacteria Found in 35 Cases of Retained Afterbirth—Metritis

	Clinical History	Streptococci	Staphylococci	Colon Bacilli	Bacilli Abortus	Bacilli Pyogenes
11 Acute cases Metritis	From 1 to 3 days	9	8	7	3	1
3 Subacute cases Metritis	From 1 to 3 weeks	2	3	2	0	2
21 Chronic cases Metritis	After 3 weeks	11	17	19	0	2
35 Total		22	28	28	3	5

A common 'bad" source of badly infected milk.

common sense figures, the profits are for the clean, healthy, high producing cow and the deficit always against the cow with a spoiled udder.

3. The dairyman profits from the physical examination of his cows by saving in time and expense of medicine for treating cows with diseased udders and also saves himself much worry.

If the physical examination of dairy cows did the consumer no good, it is worth while and will pay the dairyman a handsome profit to have it done.

However, the greatest advantage from the examination is the production of a cleaner and more wholesome milk for human consumption, especially for infants and children. Health boards and city authorities are overlooking one of the most beneficial of health regulations for milk production if the physical examination of the cow is not required in the milk ordinances of their cities or towns.

Prolapse of the Vagina in Cows[1]

By T. H. FERGUSON, Lake Geneva, Wisconsin

PROLAPSE of the vagina in cows is a condition met with quite frequently in districts where dairy or beef cattle are extensively raised.

It occurs during advanced pregnancy, and following parturition; in nymphomanic cows and frequently during or following estrum. Heifers are rarely affected. I have met with a few cases in heifers shortly before calving.

Anatomical Peculiarities Which Favor Vaginal Prolapse

The vagina of the cow is more readily pushed backward than that of any other animal because its broad ligaments are attached so posteriorily that they readily permit a posterior displacement of the vagina. Furthermore, the vagina is quite loosely attached by the pelvic connective tissue, and its greater part is covered with peritoneum in such a way as to allow more freedom of movement in any direction than occurs in other animals.

Causes

Infection of the vagina or uterus, or both, and ovarian troubles are the essential causes of the most troublesome cases of vaginal prolapse; high feeding, close stabling, and lack of exercise predispose to this condition; in fact, it is seldom seen except in cattle so kept. Uncomfortable stalls, short stalls, or those that slope so much that animals stand or lie with the posterior parts so low as to increase the intra-pelvic pressure are contributive factors in prolapse of the vagina. The intra-abdominal pressure of advanced pregnancy, bloat, bulky diet, hydrops amnii and the relaxed condition of the pelvic organs following calving, are also factors to be considered.

Infiltration of the walls of the vagina and surrounding connective tissue at the time of parturition are predisposing conditions. Coughing, urination and defecation aggravate it.

Symptoms

If the prolapse is just starting during advanced pregnancy there may be noticed, when the animal is lying down or during the acts of coughing, urination or defecation, the protrusion of a reddish mass through the lips of the vulva, which has a tendency to disappear while the animal is standing at ease.

As time goes on, this mass protrudes farther and becomes larger unless the case is handled properly from the start. Usually when the veterinarian gets the case, the following symptoms are in evidence: The animal is standing with her back arched straining more or less and there protrudes through the lips of the vulva a large red, tumor-like, mass, swollen and tense, it may have become soiled by litter or manure and it may be dark in color or bleeding from tail friction or other lacerations. The history will probably reveal that the animal has had a slight trouble for some time, is past due to calve, and the aggravated state has only existed a short time. A careful examination will usually reveal that the greater part of the vagina including the everted bladder, filled with urine, has prolapsed.

Cases following parturition are usually ushered in by violent straining. There may or may not be a retension of the placenta. The animal is apparently suffering from intense irritation of the vagina and uterus and with each expulsive effort the vagina and often the vaginal portion of the uterus is expelled through the lips of the vulva. At first, the mass partly returns to place between strains, later it becomes forced out and swollen to such an extent that its replacement is difficult.

[1] Vet. Med., Vol. XVIII, No. IV.

Cases that occur in nymphomanic cows and cows during estrum have a somewhat smaller, red, tumor-like mass parting the lips of the vulva or protruding through them. If noticed early the mucous membrane may be just a little congested, or it may be covered with a catarrhal exudate, later to become thickened and dark from exposure to the sun, insects, injuries, etc.

Treatment

Handling prolapse of the vagina taxes the skill and judgment of the veterinarian about as much as any disease he is called upon to treat. To successfully cope with this condition, our efforts should be directed at the underlying cause of each individual case. If the animal is heavy with calf, her stomachs full of bulky food, and she is confined in a stall that favors intra-pelvic pressure, and her vagina has just started to prolapse, the following treatment will take care of her case, providing her vagina or uterus are not extensively infected:

Have her placed in a comfortable stall with a level floor or one that the floor inclines slightly toward the front. Empty the stomach and intestinal tract by a very restricted diet, reduce bulky food to a minimum, give a small amount of laxative concentrated food, drinking water several times daily, no large amount at any one time. If especially indicated, to assist in quickly reducing intra-abdominal pressure give repeated, small doses of quick acting cathartics until results are obtained, or one large dose of a saline cathartic. Thereafter, constipation should be prevented by daily doses of mineral oil. This will keep the intestinal canal lubricated and thus prevent straining during defecation.

Give medicinal doses of strychnin or nux-vomica twice daily and have the animal exercised on level ground. This invigorates the patient and tends to keep the organs in their proper relations.

Cleansing and Replacement

Remove all dirt and thoroughly clean the protruding mass by flooding it with clean warm saline solution. Use a cloth to assist in removing the filth that has become dried on it. Remove shreds of mucous membrane with the scissors; dust its entire surface with equal parts of iodoform and boric acid; cover with clean cloth; and replace carefully. See that the bladder is emptied; it may be necessary to pass a catheter. Douche the vagina with a non-irritating antiseptic solution such as a 5% solution of therapogen; 2% solution of phenol; 2% or 3% alum solution; 1% permanganate solution for five minutes to as much time as is required to ally the irritation and cause complete replacement of the organs.

Drain off the fluid and smear the entire mucous surface with iodoform, subnitrate of bismuth, of each one ounce, and neutral oil one pint. If irritation is extreme spray the mucous membrane of the vagina and vaginal portion of the uterus with a local anesthetic. To assist in preventing recurrence we have made it a practice to suture the vulva in all cases, and have found it much better than any other method.

Method of Suturing the Vulva

With a sharp heavy needle threaded with a long thick waxed suture, begin on the right side of the vulva on a level with the superior commissure laterally to the delicate skin. Lift a fold of the thick skin and thrust the needle through from below upward, cross over the vulva, lift up the thick skin on the other side and thrust the needle through from above downward. Repeat this process until enough sutures are inserted to cover the vulva, tie the ends in a square knot so they may be easily released or tightened to regulate the tension. It is only in exceptional cases that it is necessary to draw the sutures taut, and as it is often necessary to leave them in for some time, the looser they are, the better. Apply a 1 to 1,000 solution of bichlorid of mercury often enough to keep the sutures and surrounding parts disinfected and clean. If it becomes apparent that the

vagina needs further treatment, loosen the sutures and douche with the warm antiseptic solution previously recommended, and follow with the iodoform paste.

Handling Serious Pre-parturient Cases

Unfortunately all of the pre-parturient cases are not so mild. The animal may be down, partly paralyzed, lying on its side usually bloated and straining more or less with a large red tumor-like mass protruding through the vulva. The mucous membrane of the mass may be dark, bleeding and covered with filth, in such a case attempt to relieve the bloat by passing the stomach tube per os, and administer through the tube, 10 drams of chloral hydrate dissolved in a quart of warm water. Slide the animal on a door or stone boat and block it up behind so that gravity will favor replacement; clean, replace and treat as recommended above and suture the vulva.

The animal should be left lying on its sternum with the posterior parts elevated. In this position she may doze for a few hours and thus obtain considerable relief. If not relieved, and parturition is about due, it may be necessary to hasten that act by attempting to dilate the os-uteri and carefully assisting the delivery of the calf. Usually in such cases, the uterus is practically inert and a dose or two of pituritin will be necessary to cause uterine contractions.

Proceed slowly with the delivery and use plenty of lubricants as the vagina is easily torn in these cases and unless care is taken it may rupture. The after-treatment includes removal of the placenta, and treatment of the uterus and vagina until they are normal. It is usually necessary to replace the sutures for a time. In cases of otherwise uncontrollable straining, the insertion of a trachea-tube will help.

Treatment of Post-Parturient Cases

The treatment of post-parturient prolapse is essentially the same as for pre-parturient, with the addition of a thorough treatment of the uterus, if the placenta is retained remove it if this can be done without injury to the uterus. If not, treat with preservatives until it comes away, or can be removed. As the irritation in most of such cases is considerable, in order to get favorable results, it is necessary to give them frequent treatments.

A common enough sequel to preparturient prolapse of the vagina.

The treatment of prolapse due to a chronic irritation of the genital organs caused by diseased ovaries consists of endeavoring to bring about a normal condition by irrigating the uterus with antiseptic solutions, draining, and injecting sufficient quantities of the iodoform, bismuth and oil preparations, and of treating the ovaries by rupturing cysts and administering pituritrin alternated with adrenalin chlorid solution. If a cure of the ovarian trouble is not successful, spaying will cause the prolapse to disappear in a short time and the animal can be fattened for beef. If but one ovary is affected, the removal of the diseased one will possibly cure the trouble and save the animal as a breeder.

Changes In Position of the Uterus

With special reference to Prolapse with Inversion
By H. E. KINGMAN, Ft. Collins, Colorado
Department of Surgery, Colorado Agricultural College

THE uterus may be encountered in any of the following abnormal positions:

1. Hernia of the uterus through abdominal wall.
2. Ventral version.
3. Dorsal version.
4. Lateral version.
5. A uterus pushed into pelvis (without inversion but with prolapse of cervical portion and vagina).
6. Inversion without prolapse.
7. Prolapse with inversion.
8. Torsion.

Some of the foregoing malpositions are very rarely met with while others are found frequently.

Inversion and Prolapse of the Uterus

Definition: the turning inside out of a part or all of the uterus accompanied by the escape of a portion, of all of the uterus from the body through the vulva. The process of inversion may begin at the end of the horn or at the vulva and vagina. Usually only one horn is inverted. The **nongravid** horn escapes, without inversion, within the inverted horn. It is perfectly possible for both horns to become inverted and prolapsed but the difference in size is one factor at least that tends to prevent inversion of the small horn.

The ovaries and broad ligaments and vessels are stretched and drawn into the serous lined sac formed by the prolapsed organ.

The ovary belonging to the inverted horn may be drawn well out of the pelvis while the other ovary retains a position within the pelvic cavity. One can picture a cross section of the prolapsed mass as being a jumble of serous and mucous and muscular layers with an interweaving of veins, arteries, lymph vessels, fat, ligaments and nerves.

The causes that directly or indirectly bring about inversion and prolapse of the uterus are varied.

Of course failure to involute is the actual cause, since the process could not take place except in a flaccid, dilated organ. But sometimes it follows immediately the delivery of the fetus especially if the expulsion of the latter is difficult and requires traction. In such cases there undoubtedly is a predisposing factor, in that, there probably exists a paretic wall favoring the occurrence of inversion and prolapse.

Intra-abdominal pressure constitutes a primary factor. That pressure may be due to labor. It is clear that labor forces the abdominal viscera against the uterus, squeezing it into the pelvis, and unless the uterus is active in its contractions, inversion and prolapse occur.

Intra-abdominal pressure may result from overloaded stomach and intestines, from excessive fat, or even tympany.

Attitude or position of the puerperal cow may be responsible for the uterus gaining a position in the pelvis that makes it susceptible to pressure and expulsive force. Further its presence as a mass in the pelvis tends to excite expulsive contraction (the afferent nerve endings that send in messages to strain, lie in the pelvic region. This is true of the bowel as well as the genital tract).

Cows that are stanchioned on floors that slope backward are exposed to a constant pressure that may culminate in the expulsion of the uterus during or immediately following parturition.

Cows pastured on uneven ground (mountain pastures) prefer to graze uphill since the food is easier to reach and

it also relieves pressure upon the diaphragm facilitating breathing, but the position throws the viscera into or against the pelvis preparing the way for prolapse of the uterus. (Prolapse of the cervix is very common prior to parturition.) It is a common practice to suture the lips of the vulva several weeks before the end of gestation in order to prevent prolapse of cervix and vagina. Emaciated subjects are more susceptible to prolapse than those in good condition. Attention has already been directed toward the relationship of involution and inversion.

The most common cause of a paretic uterus is endometritis, and the most common cause of endometritis is the Bang organism.

Many other factors influence the tone of the uterus such as emaciation or perhaps obesity.

Age has a telling effect upon the uterine walls especially in those animals that have given birth to a number of calves.

Retained placental membranes are the result of endometritis but their failure to escape brings about straining as well as traction upon the uterus. Cervicitis causes straining that is dangerous and in case the cervix remains open may permit inversion and prolapse.

The circumstances surrounding prolapse and inversion of the uterus, the condition of the patient and the disgusting and alarming features encountered are unbelievable except among those who have had occasion to deal with such cases (veterinarians).

The time that has elapsed between the occurrence of the prolapse and its discovery is one factor governing the condition of the uterus. It is unavoidably, always, covered with feces. If it has been exposed for any length of time the feces mix with the mucous covering the uterus and then dry out, making a green layer or crust that really forms a part of the mucous membrane and can not be removed without injury to the superficial coat.

The animal lies down in an effort to relieve the pain and covers the prolapsed organ with all sorts of debris (manure, straw, dirt, sticks, etc.).

In turning around the uterus may come in contact with walls of the stall, or posts, or wire of the corral, or nails of the fence. It is exposed to injury from other animals, the bites of hogs, dogs, horses, chickens, magpies (or even predatory animals). It is exposed to horn thrust, and while the cow is recumbent, to treads from other animals.

A case was encountered in a cow shipped with others in a stock car. The floor was covered with six inches of sand thoroughly mixed with urine and feces. The cow was unable to rise. The uterus was exposed for hours to the trampling feet of the other cows. It requires no stretch of imagination to picture the condition of that uterus. It was a horrible mass, punctured full of holes and filled with dirt. She died.

The fetal membranes may remain attached to the inverted and prolapse organ.

Hemorrhage from wounds or cotyledons is of more or less severity, usually it does not amount to much but occasionally the cow dies from loss of blood.

The pendent position of the uterus favors the accumulation of venous blood, lymph, and peritoneal fluids. This materially increases its size interfering with reposition, and lowers its resistance. The tissues are also tensed rendering them more susceptible to tears and other injuries. Eventually circulation is interrupted and gangrene results.

There exists no protection against whatever object large or small comes in its way. It may be sunburned or frozen.

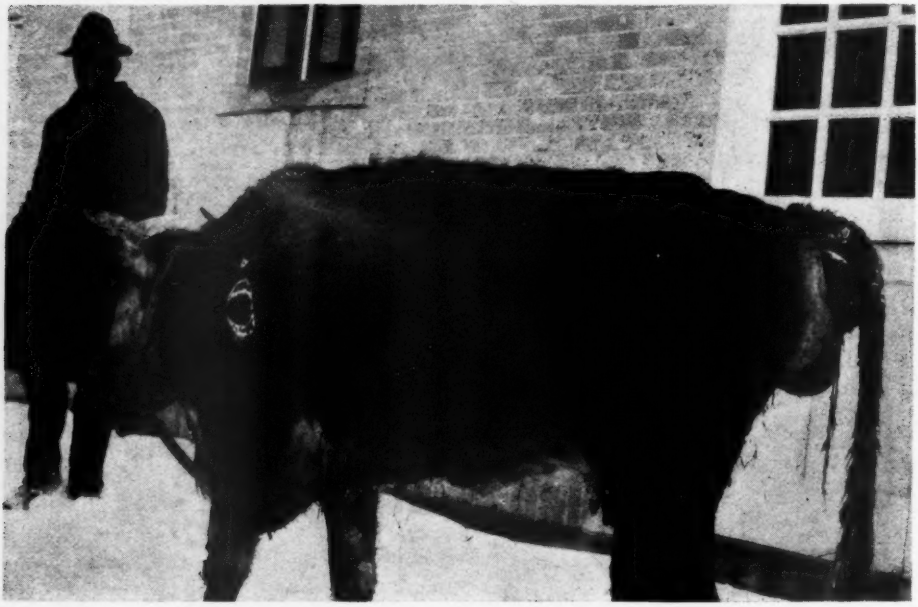
It may be soaked in the mire of a bog or drain, or scratched by a barbed wire fence.

One might go on at great length describing cases of this sordid condition.

The cautious and experienced veterinarian keeps in mind always the possibil-

ity of such an occurrence and takes every precaution to forestall it. After delivery of a fetus or removal of placental membranes he examines the uterus and corrects any malposition or any irregularity he may find. He also attends to wounds or other injuries, and leaves the uterus in as healthy and normal condition as possible. He also instructs the owner relative to the proper after care of the cow and informs him relative to the possibilities of prolapse under improper attention.

The strange thing, the unaccountable part of the whole matter is that most cases of prolapse of the uterus recover under treatment.



Prolapse of the cervix, vagina and rectum prior to parturition.

Two fundamental principles stand out as being most important in replacing a prolapsed inverted uterus. First, anesthesia; and second, position of the animal.

Epidural anesthesia given according to the technic of Benesh is peculiarly suited to this operation. All straining ceases, no resistance remains to interfere with the operator's efforts. It constitutes one of the most valuable contributions ever made to veterinary surgery.

If for any reason epidural anesthesia can not be used then chloral by the mouth or chloroform by inhalation serves to quiet expulsive straining and relieves pain.

Preparation of Uterus Prior to Reposition

If the cow is recumbent then place a canvas or sheet under the uterus to keep it off the ground.

Wash the entire organ with warm water and castile soap. (No disinfectant nor irritant soap.)

Remove carefully all debris that can be loosened without injury to the mucous membrane. If dirt is so firmly fixed on the surface that it will not come off, then allow it to remain depending upon disinfection with mercurochrome to control serious infection.

After a thorough cleansing spray the entire surface with a 2% mercurochrome

solution applied by means of an atomizer.

Cover the whole organ with a dusting of bismuth sub-carbonate or sub-gallate.

The last step in preparing the uterus is to protect it and lubricate it thoroughly with "petroil."

Elevate the hind quarters by any means practical. Probably the best way consists of a block and tackle a short single tree and two short loops of rope to place above the hocks. The legs should be raised until all viscera fall forward but the shoulders should still remain flat upon the ground.

If the uterus has been exposed only a short time and edema is not extensive, then reposition is simple. But venous and active congestion accompanied by accumulations of lymph can so engorge the organ that reposition becomes difficult and very tedious and even impossible. Of course during the handling of refractory cases the danger of injury to the uterus becomes more acute in proportion to the difficulty encountered.

The engorged organ may be too large to enter the pelvis. It then requires patience and time and skill to carefully reduce its size through appropriate massage while the uterus is held in an upright position to encourage drainage.

A 4-inch-wide rubber band wrapped gently around and around the mass helps a little to reduce its size and protect it from injury by the fingers, it also makes the uterus easier to handle.

One begins at the vulva to return in the wall of the inverted organ. The whole mass is pushed into the pelvis with flat hands. As one succeeds with the whole circumference, an effort is made to work the lips of the vulva back over the mass, tucking in one cotyledon after another, carefully and patiently exercising judicious massage and pressure, until finally the entire organ disappears through the lips of the vulva. At this stage of the operation a repositor is useful.

Ordinarily involution begins immediately rendering a recurrence unlikely but in order to assure its permanency the vulva is closed by heavy sutures always carried back to the hair line. Quills are used to support the skin and two inch muslin bandages are used for suturing material and left *in situ* 48 to 72 hours.

The prognosis is favorable providing the uterus has remained intact; even small holes through the dorsal wall are not always fatal.

Neither is it always necessary to be particular about the cleansing of the mass. I know of case that occurred "on the trail." The cowboy roped the cow, replaced the uterus without either washing his arms or the inverted, dirt covered, mass; closed the vulva with wire from a fence and turned the cow with the herd.

The good fortune following such cases is difficult to explain. It should not be taken as an excuse for neglect on the part of the veterinarian or failure to handle his cases with all the skill and efficient tenderness that the case requires.

RUPTURE OF THE REPUBIC TENDON.

Diseases of the Udder

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THE subject "disease of the udder" is so comprehensive that I can only discuss certain clinical aspects in the space allotted. The real reason for such a discussion perhaps, lies in the fact that even though much valuable material has been written concerning all phases of this subject, we all realize the hopelessness of the task of attempting to live up to the expectations of many owners of cattle when called upon to treat certain of these diseased conditions. It seems to me that veterinarians in general appreciate the technical side of the question quite well. However, the utter lack of a proper understanding of owners and herdsmen concerning the delicate complexity of structure, the vulnerability of the udder, and the improbability of complete recovery from even moderately severe infections, clouds the issue and very materially complicates conditions for the clinician.

Careful technical supervision of milk production and immediate professional intervention in case of the slightest evidence of disease are highly important if not necessary considerations for both the production of wholesome milk and the successful control or treatment of udder diseases. Infections of the udder are not specific contagious, infectious diseases but are comparable to any local wound infection.

Whether the condition be a hard milker, a tumor or an injury, aseptic or complicated by an infection, the problem is largely surgical in nature and requires the most rigid application of the fundamental principles of surgery. While more or less careless surgical technic may not be attended by appreciable ill effects when applied to some regions of the body, we can readily undertand why such methods fail miserably when applied to the udder, if we carefully consider the minute and gross structure of the organ, its function and economic importance.

The importance of a thorough knowledge of the anatomy and physiology of the udder cannot be overstressed if we are to give proper advice concerning the care of the udder and expect to intelligently diagnose, prognose and treat the diseases affecting this organ. Perhaps there are some anatomical and physiological features of special significance to the clinician that should be summarized in this paper.

1. F. T. Harvey, F. R. C. V. S. (*N. A. Vet.*, Mar. 1923, 130) calls attention to a very practical point, viz: "In most glands doing a large amount of work the secretion as it is formed is carried away or stored at a safe distance, as seen with the urine and bile, and infection of the contents of either of these cisterns may leave the working cells of the associated glands quite unimpaired for long periods. But there is no protected or guarded route between the galactophorous sinuses and the gland acini, so that the parenchyma of the organ becomes quickly involved in any bacterial pollution of the milk in the receptacles."

2. Physiologic resistance to infection. A significant fact was emphasized by Dr. F. S. Jones (*Jour. A. V. M. A.*, Dec. 1927, 354), at the Philadelphia meeting in 1927, viz: "In regard to resistance to udder infection, nature has given two substances that we may speak of as factors in natural resistance. The first is the bactericidal property of cows' milk. If you take fresh, raw milk not heated above 143 deg. F. and innoculate it with a mastitis streptococcus, growth does not take place within four to eight hours. If you boil the milk, growth begins at once and in a few hours the plates are overwhelmed. This bactericidal activity of milk is one point. Unfortunately, the dairyman under modern circumstances has rendered this well-nigh inoperative. The period of growth inhibition rarely runs over eight hours, and unfortu-

nately we have this purely arbitrary method of milking twice a day, which has rendered the inhibition more or less inoperative." As a second factor of natural resistance Jones states that "The mastitis streptococcus will not grow in the blood serum of cows." D'heil (*Jour. of P. & T.* 19, 51), says that the gland tissue of the udder possesses a strong bactericidal power.

3. D'heil also states that the teat canal and milk cisterns generally contain bacteria which gain entrance through the teat canals and moreover the gland tissue of the udder contains bacteria though in small numbers. Within so called normal limits these passive residents seem to be harmless, attenuated and non-pathogenic, though under favorable circumstances may become virulent. The first jet of milk drawn always contains the greatest number of bacteria and is therefore discarded in the production of certified milk.

4. Harvey (*N. A. V.*, April, 1923, p. 186) quotes Irmindorf to the effect that milk from healthy cows contains 1 to 5 cc. of leucocytes per liter of milk. And furthermore that any appreciable increase in the leucocytic count is generally associated with the presence of numerous streptococci.

5. The teat sphincter assists in maintaining surface contact of the mucosa lining in the teat canal and thereby forms a relatively efficient barrier to the entrance of harmful amounts of infection under ordinary circumstances.

6. Emmerson [1] has shown that the principal artery to the teat is located in the lateral wall.

7. Milk that has lost its bactericidal property becomes excellent media for bacterial growth.

From such knowledge we can readily picture the possible results from lowering the natural resistance of the organ or protective powers of the milk by certain predisposing factors or from the direct introduction of either mass infection or an infection that is especially virulent. Our most serious diseases of the udder are either primarily or secondarily due to the effects of bacterial invasion. Likewise most of our teat obstructions, so called "spiders" or teat tumors, thickenings of the mucosa and submucosa, and the membrane or veil of tissue that completely separates the base of the teat from the milk cistern, are all products of more or less chronic catarrhal inflammation.

We all realize the importance of a thorough knowledge of the so called predisposing factors concerned in diseases of the udder both from the standpoint of prevention and treatment. I shall briefly comment upon these factors.

1. Insanitary conditions cannot be defended nor tolerated in the production of wholesome milk and are quite a factor in diseases of the udder. The premises, milkers, milking machines and cattle should be clean and free from dangerous contamination such as wound discharges or contagious disease. The udders and teats should be clean and dry at time of milking.

2. Irregular, rough or incomplete milking. Intentional failure to milk for the purpose of "bagging up" the cow for sale is a vicious practice. Cows should be milked regularly and thoroughly (and it would seem that eight hour intervals might be preferable to milking twice daily as is generally practiced). Care should be exercised in drying up the milk secretion.

3. Cold—chilling the udder by permitting it to become or remain wet in cold weather or by lying on cold damp floors, etc.

4. Bruising or wounding the udder or teat. Fast, hard driving, rough treatment, severe bunting by the calf, stepping on the teat, wire cuts, etc.

5. Forced feeding for high milk production. Klein (*Jour. A. V. M. A.*, Oct. 1922, p. 30) reports observing attacks of catarrhal mastitis in certain cows whenever their grain ration was increased beyond a certain limit.

6. Keeping cows with infected udders, either actively diseased or passive carriers of pathogenic strains of bacteria, in the milking line.

[1] Dr. M. A. Emmerson, "Anatomy of the Udder of the Cow," unpublished thesis, Library, Iowa State College, Ames, Iowa, 1928.

7. Keeping cows with abnormal vaginal discharges in the milking line.

8. Milking the contents of infected quarters on the floor. Such material should be milked into a vessel containing a potent disinfectant and then properly disposed of.

9. Precocious secreting of milk by young heifers induced by calves nursing at the teats.

10. Cow pock—sore teats or udders should be given prompt attention and precautions taken to avoid serious complications.

11. The use of milk tubes for hard milkers, injured teats, milk fever, etc., is safe only when proper precautions have been taken. This means that every step should be as carefully planned and executed as though the surgeon were opening the abdominal cavity.

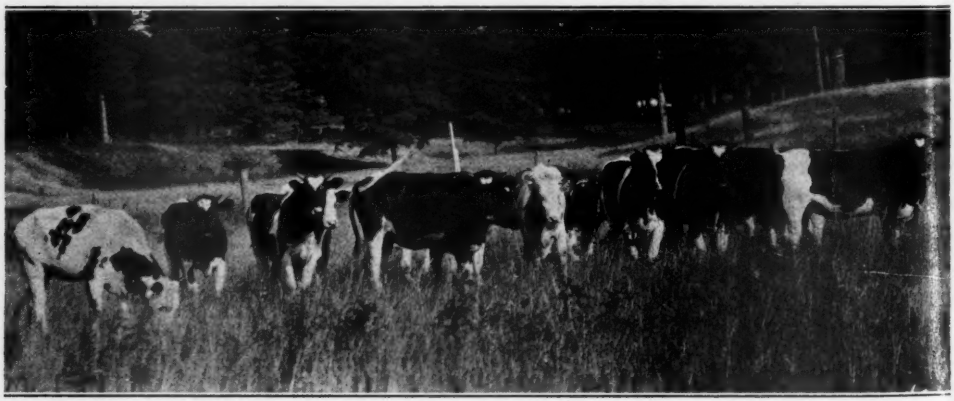
12. The Bang bacillus is incriminated by some authors as a factor in lowering the resistance of the udder to other infections. It is also a fact that where contagious abortion is present in a herd there also exists a higher percentage of metritis and abnormal vaginal discharges.

LYMPHATIC EDEMA—CAKED UDDER

This is not purely speaking a pathological condition since it is quite generally considered as merely indicative of high milk production. It is most frequently observed in young cattle just prior to first or second parturition. It is usually classed as a border line disease. In such a condition the udder is very easily injured, thus lowering the resistance of the organ and causing it to be very receptive to infections. The prognosis is usually good; many such cases are not treated. Avoid high protein diet prior to parturition, running or long hard drives or any unnecessary exposure of the udder to injury. Some recommend a "cold shower" in the early stages or later application of heat and massage. Dry the surface of the udder thoroughly and apply a non-irritating ointment or fresh lard to protect the surface and facilitate massage.

Turner (*Jour. A. V. M. A.*, Dec. 1927, p. 352) concludes after a limited trial that "the use of streptococcus bacterin prior to parturition, has been highly successful in preventing the development of those enormously distended udders occurring so frequently among high producers, which frequently develop mastitis." A herd autogenous bacterin was used during the drying off period—five cubic centimeters or ten thousand million organisms was administered every three days until six doses were given. He states, however, that about four months after vaccination 10% of these cows developed mastitis while only 13% of a much larger group that were not vaccinated developed the same trouble.

Ames, Iowa. W. F. Guard.



Mastitis in the Cow

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THE inflammatory reaction observed during an attack of mastitis in the cow is merely a natural curative process designed to overcome the invading irritant and to repair the damaged tissue.

Aside from the predisposing factors, the irritant is most commonly bacteria and their toxins, and the symptoms vary considerably depending upon the specific type of organism present, and its virulency as well as the location and extent of the irritant. Various strains of streptococci, both hemolytic and non-hemolytic have been associated with mastitis and their prevalence over other types of infection is generally recognized by veterinary pathologists. Staphylococci are occasionally incriminated either alone or in conjunction with streptococcic infection. In the early stages of acute mastitis it is seldom if ever possible to differentiate clinically between streptococcic and staphylococcic infection. A bacteriological examination is therefore essential especially if we have a herd problem or contemplate the use of vaccine, bacterin or antisera. According to Wall (Mastitis of the Cow, Crocker) streptococci, staphylococci and *B. coli* cause parenchymatous or milk infection and only stroma infection of an acute character while *B. pyogenes,* tubercle bacilli, actinomyces fungi and the necrosis bacillus produce typical stroma infection of a chronic character.

Classification

Depending upon the predominating local symptoms we recognize clinically three types of mastitis, viz.: catarrhal, parenchymatous and interstitial. It is doubtful if it is possible to have any one of these types in a severe form without having one or both of the other types as a complication. This anatomical classification should not confuse us regarding the true nature of udder infections.

Since it is generally believed that most of the udder infections occur through the teat canal it is reasonable to believe that the majority of mastitis cases are primarily catarrhal mastitis. It is also obvious that the earliest possible recognition of this condition is essential to the successful control and treatment of mastitis.

Catarrhal Mastitis

Catarrhal mastitis is essentially an inflammation of the mucous membrane lining the teat canal, milk cisterns and milk ducts. This does not necessarily mean that the entire mucous membrane lining these structures is always involved. In fact clinical and pathological evidence often indicate that mild, subacute or chronic inflammations may exist and remain quite localized especially in the teat canal. Thus the local proliferative changes affecting the mucosa and sub-mucosa often noticed for the first time when the cow freshens. There is no history of a previous attack of mastitis and such conditions are undoubtedly due to a rather localized attack of catarrhal mastitis probably induced by improper methods of "drying up" the milk secretion or to some other local contributing factor. However, we should not lose sight of the more probable and dangerous course during lactation, viz.: an extension of the inflammation to the parenchymatous cells or interstitial tissue unless prompt recovery is induced.

Symptoms, Course, and Pathology. Klein (*Jour. A. V. M. A.,* Oct. 1922, p. 26), has undoubtedly recorded the most careful and extensive observation on this type of mastitis and I shall quote him on the symptoms, course and pathology.

In two-thirds of the cases of catarrhal mastitis there is no swelling of the udder apparent at the beginning of the disease, and the other third shows only slight swelling, with little heat and not much pain. Generally only one-quarter of the udder is affected.

One of the first symptoms observed is the presence of small white flakes in the first few streams of milk from the affected quarter. These flakes are often quite small, frequently no

larger than a pin head, and may not be very numerous. Unless the milk is drawn into the palm of the hand or on a finely meshed wire strainer and closely examined, they will not be noticed. In ordinary milking they are almost certain to escape detection. Milk from a quarter which is not diseased may show a flake or two now and then but this does not happen often and does not occur at successive milkings.

Another early symptom is difficulty in expressing the first stream or two of milk. It is usually stated that the cow "milks hard," although formerly an "easy" or "ordinary milker." Sometimes it is reported that the cow kicks when milking is begun, whereas formerly it submitted quietly to the process. These symptoms are due to obstruction of the teat canal resulting from swelling of the mucous membrane or collection of exudate. The stream of milk expressed from the teat may be split or deflected from the normal direction when particles of exudate collect and dry around the outlet of the teat canal. If the end of the teat is examined in such cases before milk is expressed, small yellow crusts will be seen around the orifice of the teat canal. Sometimes, however, dried crusts of milk collect at the same point and change the direction of the stream in a similar manner when no disease is present, probably because of fatigue of the sphincter between the lower end of the milk cistern and the upper end of the teat canal. Splitting or deflection of the first few streams of milk, in the absence of any other changes, is therefore not necessarily an indication of the presence of catarrhal mastitis.

COW SUFFERING FROM ACUTE STREPTOCOCCIC MAMMITIS
Note the intense edema of the udder and under the abdomen.

Another of the early symptoms is that the stream of milk is not cut off completely when pressure is removed from the teat, with the result that the end of the teat is smeared with milk. This is caused by the mucous membrane being swollen and interfering with the closing of the sphincter. In the early stages of the disease, a drop of mucus or pus may be squeezed out of the teat canal by pressure on the end of the teat.

While catarrhal mastitis is primarily an inflammation of the mucous membrane of the large milk canals, milk cistern and teat canal, the inflammation, if not checked, extends into the submucous connective tissue, producing proliferative changes. In this way the mucous membrane of the milk cistern becomes thickened and may then be felt as a hard cord about as thick as a lead pencil running through the middle of the teat when the teat is rolled between the thumb and fingers. Flat disc-shaped thickenings and nodular indurations may be felt in the upper limits of the milk cistern around the terminations of the large milk canals.

Growths appear upon the wall of the milk cistern and teat canal as a result of hyperplasia of the epithelium and may be detected by palpation. These often interfere with the withdrawal of the milk. This particular condition is known in some dairy sections as "spider in the teat." The proliferative process, when once started in the submucous connective tissue, may extend upward into the interstitial connective tissue of the glandular part of the udder, producing indurated areas of greater or less extent, and the newly formed connective tissue, subsequently contracting, causes atrophy of the gland cells with permanent loss of function in the area affected. The entire quarter may be involved, in which case the cow becomes a "three-teater."

Catarrhal mastitis does not affect the secreting cells of the udder, and therefore the milk does not show any marked changes, at least not in the first stages of the disease. It has been mentioned that the appearance of small white flakes in the milk is one of the first indications of catarrhal mastitis. These flakes are usually present in the first few streams from the teat of the affected quarter, but sometimes they appear in the middle or at the end of the milking. But to the casual observer, the secretion at this time has the general appearance of normal milk and throughout the entire course of mild cases may not show any marked change.

As the disease progresses, however, the careful observer will detect clumps of mucus or pus, usually in the first few streams of milk, or the first expression may be entirely pus and followed by milk of normal appearance. The first few streams from the teat may gradually assume more and more the appearance of pus, but the secretion subsequently drawn from the udder will have the appearance of normal milk unless the disease extends upward into the secreting structures, and then a turbid flow resembling whey, or a fluid resembling serum, containing clots of fibrin and clumps of pus, will be obtained from the affected quarter.

In cases of severe mucous catarrh the secretion from the affected quarter becomes graddually more and more slimy and viscid and takes on a grayish tint. By carefully observing the changes in the secretion the extent and character of the disease may be judged, and these changes are also of assistance in making a prognosis. When the whey-like or serum-like fluid is present or the secretion contains much pus, the prognosis is unfavorable.

Jones (*Jour. A. V. M. A.,* Dec. 1927, p. 354), says, "A good many cows in heavily infected dairies carry streptococci in the udder and show no symptoms. Those carriers fall into three groups: First, the cows in the stages of incubation; second, those that have recovered from an attack of mastitis and have not entirely eliminated the streptococci from the udder; and a third group which carries the organism and yet has never shown clinical symptoms of the disease." Practically all authors agree that streptococci are the most common offenders but that staphylococci are occasionally responsible or involved.

Parenchymatous Mastitis

This is a very common complication of catarrhal mastitis though it is often secondary to interstitial mastitis or may develop quite independently and spread to the milk ducts and interstitial tissue.

The secreting cells are involved and we have the invading forces fighting against the local and mobilized forces, producing irritation, cell injury or destruction.

Symptoms.—The symptoms naturally vary within wide limits depending upon the severity and extent of the attack and the resistance of the udder and the cow. In the acute form the gland tissue becomes swollen, tense and sensitive to touch and the milk secretion becomes reduced and thick and pus like. There may be an early rise of temperature and the animal may have a tendency to chill. The pulse varies considerably according to the general reaction. The appetite is capricious and there is a tendency toward constipation. The feces are dark colored and often covered with mucous. In the latter stages of severe attacks, there is diarrhea, the feces being thin, and having a very offensive odor. In these severe attacks any abating of the general symptoms with localization and abscess formation is usually considered favorable for the cow but at considerable expense of the gland tissue involved.

In the chronic form of this disease the symptoms are less severe and extend over a long period of time with a shrinking of the quarter and eventually perhaps complete disfunction.

Interstitial Mastitis

This is an inflammatory process which preeminently involves the connective tissue or supporting structure of the udder. It is a phlegmon and the symptoms are much the same as phlegmons located elsewhere. The course may be acute and involve one quarter or the entire gland, as where a case of lymphatic edema becomes complicated by infection, and may result in necrosis of the gland tissue and perhaps death of the cow if not given early assistance. Single or multiple abscesses often develop. Or it may run a more or less chronic course with proliferation and induration resulting in atrophy of the gland tissue.

The infection may enter through any external wound or from parenchymatous necrosis. It often originates from septic inflammations of the skin or mucous membrane of the teat extending to the deeper connective tissue elements and thence to the interstitial tissue. Such reactions quite readily spread to other quarters since the connective tissue of one quarter is continuous with that of others.

Mastitis sometimes spreads rather rapidly in a herd and it is sometimes difficult to determine the exact method by which it is carried from one animal to another. Antiseptics are not a successful substitute for cleanliness and faith in them merely creates a false sense of security. Where milking machines are used it is sometimes the practice of herdsmen to depend upon B. K. or some other chlorinated lime solution or antiseptic for cleansing the apparatus and the same solution is sometimes used indefinitely. Samples of such solution may be found to be teeming with organisms.

Symptoms.—In acute primary interstitial mastitis the process is usually local at first and presents the symptoms of any local wound infection. The infection may spread and involve the entire udder. The milk secretion is diminished but not altered much in appearance. Such a condition occasionally complicates cases of lymphatic edema. Chronic interstitial mastitis is recognized by

induration. The induration may be present in more or less localized areas or quite evenly distributed.

Treatment

The degree of success obtained in the treatment of any case of mastitis depends upon first, how soon you are called after the first symptoms of disease appear and, secondly, the effectiveness of methods used to assist the natural reaction to overcome the irritation and repair the damage.

1. Rest and conservation of energy are generally recognized as absolutely essential in fighting any infection. Clean, comfortable quarters should be provided. Encourage diminution in milk secretion by removing grain and succulent foods from the ration. Supply plenty of fresh water to drink. Both rest and protection for the udder may also be encouraged by the effective application of a suspensory bandage.

2. Encourage the removal of the stagnant exudate and encourage the supply of blood and fresh exudate to the part. This may be accomplished by:

First, frequent milking—every one or two hours. If indicated do not hesitate to slit the teat sphincter or the end of the teat to facilitate drainage.

Second, the application of moist heat and a suspensory bandage. Especially in cold weather, an antiphlogistine pack is to be preferred. This may be placed on a stove top or in an oven and reheated as often as indicated.

Third, assist the natural channels of elimination by the use of purgatives, diuretics and rectal injections.

Fourth, where the stasis is obstinate and extensive necrosis or the danger of resorptive fever apparent it will be of great assistance to resort to incisions for drainage. Splitting of a teat or its amputation are sometimes resorted to. In making incisions into the gland tissue proceed cautiously to avoid severe hemorrhage. It is best to make small openings first and enlarge them by subsequent operations. Such wounds should be thoroughly disinfected and precautions taken to avoid secondary infection entering. Abscesses of the interstitial tissue should be drained at once.

Fifth, medication. In addition to the use of purgatives and diuretics the use of formalin as suggested by J. N. Frost is generally considered to be quite practical. It is administered in one-half to one-ounce doses diluted in a quart of milk or cold water twice daily. Larger doses are apt to produce serious digestive disturbances.

Sixth, use of bacterins and vaccines. Upon this point there is considerable variation of opinion. Many practitioners use them both for prevention and treatment. Most observers agree that the use of autogenous bacterins are superior to stock bacterins for treatment and are only used where a streptococcic infection is present.

It is difficult to comprehend how preventive vaccination could be any more effective for udder infections than for ordinary wound infections located elsewhere such as arthritis, metritis, peritonitis, etc.

Jones says, "by stimulating the antibodies in the blood serum you may perhaps alter the character of the infection, but so far no one has been successful in actually preventing udder infection by the injection of vaccine." However, where one has an outbreak of mastitis in a herd it is logical to suppose that vaccination of individuals not yet infected might have a beneficial, though very temporary effect.

Carpenter (Report of the New York State Veterinary College, 1923, 1924), states that although the injections of the dead alpha-hemolytic streptococci are of apparent value for the control of bovine mastitis, that unquestionably good results have been obtained from the use of the living suspension of this organism. They shorten the course of the disease and check the resulting inflammation so that fewer tissue changes take place.

When signs of chronic induration and atrophy of the udder appear, use counter irritation. Klein recommends painting the affected part with a mixture of tincture of iodin, 2 parts, and oil of turpentine 5 parts. This is applied two or three times daily until the skin becomes tender. Tincture of iodin may be used alone.

Wounds, Fistulae and Obstructions of the Teats

By W. F. GUARD, Ames, Iowa

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Wounds

Wounds of teats and udder are quite common and may be superficial or deep. It is natural to suppose that such wounds are infected and the importance of prompt and effective care cannot be over emphasized.

1. Thoroughly cleanse all such wounds and apply tincture of iodin to further insure against infection. Tincture of iodin is used only as a primary treatment since it is too irritating to the wound tissue and skin for repeated application. Protect these wounds carefully from further irritation of any nature. A finger cot may be used advantageously on injured teats and where infection is already present you may apply antiseptic treatment in this manner.

If it is a gaping wound, it is often an advantage to apply sutures. Carefully approximate the wound edges but do not tie the sutures too tightly as the swelling will cause them to cut and irritate the tissues. The application of adhesive tape is sometimes used either alone or after sutures have been applied. It helps to immobilize the structures and gives surface protection. The end of the tape should be applied near the edge of the wound and then wrapped away from the wound and applied carefully and smoothly entirely around the teats. In this manner more accurate approximation of the wound edges can be obtained.

If a milk tube is to be used the following rules should be observed. Express one or two streams of milk to cleanse the teat canal. Thoroughly cleanse the teat and paint the end of the teat with tincture of iodin. The milk tube must be sterile when it enters the teat canal. This necessitates clean hands and careful protection and handling of the tube which is known to be sterile. A milk tube with a screw cap that can be secured in position and left several days is much preferred to repeated applications, since proper cleansing and sterilizing the teat is irritating, and multiplies the danger of introducing infection. Milk tubes should not be placed in the hands of laymen to introduce.

Where the end of the teat is badly injured it may be best to amputate it. This does not mean that the sphincter mechanism will be destroyed. The wall of the teat is composed of skin, subcutaneous connective tissue, smooth muscle fibre, submucosa and mucous membrane. At the base of the teat there is relatively much less muscle tissue and a greater amount of connective tissue while this condition gradually changes until the reverse relations exist at the end of the teat. Clinical experience indicates that perhaps three-quarters of an inch or more of the end of the teat may be removed and still have sphincter control following healing.

Subcutaneous Injuries

In bruises of the teat, the teat tissues become swollen and edematous and it is difficult to withdraw milk. The use of a milk tube in these cases is of primary importance, but it should not be inserted until the teat canal has been thoroughly cleansed. Hot applications may also be used to encourage resolution. Protect against secondary injuries and contamination.

Fistulae

Deep wounds of the teat resulting in milk fistulae must be treated within a very few hours to insure the best results. After thoroughly cleansing and disinfecting the wound and teat, a sterile milk tube is inserted and the wound is closed with silk or linen sutures. The sutures should be placed well back from the edges of the wound and extend obliquely down to the submucous tissue where the mucous membrane is open. Thereby more perfect approximation of the

wound edges is obtained. A protective dressing of adhesive tape or collodion may be applied and left for several days.

Chronic Fistulae

Chronic fistulous tracts are best treated when the animal is not lactating. The tract may be dissected out, removing an elliptical-shaped piece of tissue down to the mucous membrane. Then apply sutures and dressing as for deep wounds. If the tract is small cauterization may be used in place of operating. Prepare the teat and pass a sterile milk tube. Then with a long, fine, point heated to a cherry red cauterize the tract.

Strictures

Stricture at the external sphincter producing a so called hard milker. May be congenital or acquired. These conditions are best treated by the use of the Stoll method, (Dr. L. V. Stoll) and instrument. The operation is performed in the standing position with the head properly confined and an assistant holds the tail rigidly erect. Carefully prepare the teat by expressing a few streams of milk and painting the end with tincture of iodin. Then compress the end of the teat between the finger and thumb and with the probe point of the Stoll slitter directed into the teat canal, suddenly thrust the cutting edges just through the compressed sphincter and withdraw the instrument. Milk several times during the first 24 hours.

Polypi

Pedunculated growths may sometimes be palpated on the inside of the teat canal and interfere with milking. The cow should be properly confined and the teat thoroughly prepared for operation. The teat canal may be swabbed or irrigated with a dilute iodin solution, 10% Lugols solution, 2% tincture of iodin in glycerin or a 10% mercurochrome solution. If the teat sphincter is too tight the end of the teat may be slit. Then with the alligator forceps and scissors (introduced by Dr. Lothe) the tumor may be grasped and severed at its attachment. If the tumor is too high to permit using the forceps the scissors may be directed by careful palpation, or rollers may be used.

Thickenings with a broad base in the submucosa resemble definite growths on palpation. It has been my experience in attempting to remove these processes that they are quite indefinite and composed of a loose embryonic type of connective tissue involving the submucosa and often completely encircle the canal. Such thickenings may seem to be definite spherical enlargements in the teat canal, completely obstructing it. I find after thoroughly preparing the teat inside and out I can conveniently pass a grooved director through the center of the enlargement and by turning it a few times make sufficient opening for the passing of a Hug's teat tumor extractor. By carefully manipulating the instrument with the right hand while fixing the position of the tumor with the left hand I gradually cut away most of the thickening. Enough of the mass is removed to permit of a free flow of milk. A milk tube with a cap on it should be used to give rest and protection to the teat.

Complete obstruction of the flow of milk caused by a membrane located at the junction of the upper part of the teat canal and the milk sinus is apparently a result of a catarrhal inflammation of the mucous folds at this point. The inflammation is of a chronic type occuring during the dry period and the surfaces of the folds fuse forming a complete obstruction or bands of tissue in this region. These membranes may be thin and easily ruptured or may be quite thick and tough.

Properly confine the animal and prepare the teat and instruments for an eseptic operation. The teat canal should be swabbed with tincture of iodin or flushed with a dilute iodin solution or a 10% mecurochrome solution. The membrane may now be punctured by the use of a teat sound such as a grooved director. If your grooved director has a cutting edge near the point you may be able to cut a more or less circular piece of this membrane away. After which you may pass a blunt pointed teat bistoury to sever the membrane at several points. The sinus should be kept distended with milk for the next four or five days by only partially removing the milk every few hours.

Bovine Tuberculosis Eradication

By J. A. BARGER, Des Moines, Iowa

THE manager of a packing house in a rural section, in speaking of tuberculosis, said: "take our records for the year 1928 and picture a train of 75 carloads of hogs and 17 carloads of cattle —this is the equivalent of the number of hogs and cattle condemned and not fit for number one food—in addition to this loss there is equally as large a loss, if not greater, in the loss of primal parts such as heads, plucks, viscera, etc." What is true in the case of this small country packer is true also in the case of the larger packers who draw their cattle and hogs from a more extensively diseased area and where selection of buying can not so easily be made from areas or farms where the cattle have been tested.

Everywhere there is an increasing demand for milk from herds that are free of tuberculosis and for breeding stock from the same source. In fact, the public, by taxing itself, has arranged for the eradication of tuberculosis and provided, in this way, that those who sustained a loss of reactors can be reimbursed liberally, that they may replenish their herds and continue in business without a serious financial handicap. With this ample provision having been made it is not strange that the public expect the protection which its money was appropriated to give. Neither is it reasonable to presume that a man can continue to produce hogs or cattle from diseased dams and sires, nor can it be expected that purchases will continue to be made by the packers indiscriminately of whether or not the herd or locality from which they came harbors the disease of tuberculosis.

The elimination of waste is the one great factor that brought about the program of eradicating tuberculosis. Whether this waste be in animal flesh, or the life of man, or conserving the energies of man by preventing him from becoming a cripple or diseased from drinking or eating food contaminated with disease producing germs it is nevertheless waste and should be prevented. And all subjects relating to the eradication of tuberculosis should be approached from that angle.

The veterinarian has played a conspicuous part in the eradication of every serious malady of live stock, beginning with scab, dourine, cattle fever ticks, and foot and mouth disease. His attention now and for some time in the past has been focused upon bovine tuberculosis and the suppression of this disease will mean a tremendous saving in many respects. It is gratifying to know that the job is more than half finished. It is also gratifying to know that the disease in the human family is being rapidly reduced. In the latter case perhaps the greatest reduction has occurred in the number of crippled children as a result of bone tuberculosis.

Our part of the work naturally centers in completing the job. The Congress and the various state legislatures have not and will not hesitate to provide funds for this undertaking but they, like the tax-payers, expect results for their expeditures. Every veterinarian should interest himself in this undertaking, if for no other reason than that it is a disease control project. Those who lend their active assistance should see that appraisements of animals are not in excess of their actual values and solicit cooperation of the cattle owners to the end that a reasonable number of animals may be tested each day. Also, that the farmers with whom he comes in contact are furnished information in justification of the testing. Virtually all of the opposition manifested by the farmers is on account of lack of information from the proper source and practically all of the opposition that has come to our attention has its origin in organizations that are opposed to medication or who do not be-

lieve in the use of biologics. Anyway, every great movement, whether it is the building of roads, schools, or what not, must anticipate opposition of some sort, especially when public funds are being used. Opposition, however, to tuberculosis eradication work, has served a very good purpose for it has brought out an able defense from such men as Dr. Chas. H. Mayo (1), Dr. D. C. Lockhead (2), Dr. W. A. Evans (3), United States Senator Copeland (4), Dr. L. Van Es (5), Dr. Wm. H. Park (6), Dr. H. N. Bundesen, and scores of others who might have remained in obscurity, so far as this work is concerned, had they not become indignant over the false accusations concerning the tuberculin test. Moreover, when the statement was made that many of the reactors were not showing tuberculosis on autopsy Biester & Murray (7) proved that a high percentage of so-called no-lesion reactors which had come to their attention showed the organism of tuberculosis on laboratory investigation. This has also been confirmed by other research workers. Field investigations show approximately 70% of the no visible lesion cases came from herds where the disease of tuberculosis was known to exist. This coupled with the fact that, based on the number of cattle tested, only three animals out of every 1,000 proved to be a no-lesion case dwarfs the no-lesion proposition to almost the vanishing point and robs the objector of his most potential weapon.

I trust no one will overlook the importance of being able to furnish information to justify the necessity for testing cattle and the careful administdation of tuberculin. It is believed that the syringe used should have a capacity not in excess of 30 minims and equipped with a very small needle and a gauge to regulate the dose; that the dose not exceed two minimums; that the injection be made in the caudal fold and into the skin of the vulva or in case of male animals, into the fold of the rectum. The thought being that it is well to have two injections in all animals. In a questionnaire submitted to approximately one hundred practicing veterinarians who had used both the single and double test method in area work for a considerable length of time, only a few voted negatively to the question, "Is the Double Injection Advantageous?" All of them felt that the lower injection was more dependable in their hands than the ophthalmic test.

We should all qualify ourselves to be a factor in the suppression and eradication of tuberculosis. It means a more permanent live stock industry and a more wholesome milk supply generally.

[1] Senate Document No. 7. "The Danger to Man of Bovine and Avian Tuberculosis."

[2] "Bovine Tuberculosis in Humans" presented at the Second Annual Midwestern Tuberculosis Conference, at Omaha, Nebraska, June 29, 1926.

[3] "Bovine Tuberculosis as a Public Health Problem" presented at the 31st annual meeting of the United States Live Stock Sanitary Association, Chicago, Ill., Nov. 30, Dec. 1-2, 1927. (Reprint from Journal of the A. V. M. A., Vol. LXXII, N. S. 25, No. 6, March 1928, pp. 823-841.)

[4] "Importance of Pure Milk Supply" Midwestern States Tuberculosis Conference, Sioux Falls, South Dakota, June 5, 1929.

[5] Circular No. 23, University of Nebraska.

[6] "The Relation of Milk to Tuberculosis" excerpt from paper presented before the Fifth Conference of the International Union Against Tuberculosis, Washington, D. C., 1926.

[7] "Studies on a Gross No-Lesion Tuberculin Reactor" reprint from Journal of the A. V. M. A., Vol. LXXIII, N. S. 26, No. 7, November 1928, pp. 848-854.

PAINT POISONING IN CATTLE

The season for lead poisoning of cattle seems to be open and in full swing if the laboratory reports are any criterion. Cattle are very susceptible to lead and can be fatally poisoned with relatively small amounts. Lead in the form of paint seems to have a peculiar attraction for cattle. They will go out of their way to secure and eat it. Sign painters, house painters, barn painters and painters of other types are abroad in large numbers. If cattle die suddenly, particularly while in pasture, investigate the possibility of accessible paint.—*Michigan Veterinary News.*

Hemorrhagic Septicemia

By ADOLPH EICHHORN, Pearl River, N. Y.

HEMORRHAGIC septicemia is a collective name for those infectious diseases of different species of animals which are caused by the bacillus bipolaris septicus. Not only does the causative agent primarily attack the animal and cause outbreaks with great losses, but the germ is frequently secondarily responsible for losses among live stock. The disease may occur as an independent infection in cattle, sheep and hogs. The organisms are found also as secondary invaders in hog cholera, equine influenza (shipping fever) and in canine distemper. Recently, McGown attributes a destructive disease of sheep in Great Britain, known as braxy, to a variety of this organism.

Hemorrhagic septicemia or bipolar organisms are very widely distributed in nature. They are found in the soil, on various plants, in stagnant water and even on the normal mucous membranes of animals. Under such conditions, they apparently live as saprophytes, but under favorable environment they become pathogenic causing outbreaks of the disease, and later after the infection has been overcome they return to the harmless stage.

The disease running as it does as a true septicemia the symptoms and postmortem lesions are characteristic of such an infection. It manifests itself either in an intestinal or pectoral form with a marked febrile reaction, and not infrequently, the symptoms and even the anatomical changes resemble those of anthrax.

On postmortem examination, a subcutaneous exudate is found which is gellatinous in character. The hemorrhages are present in the connective tissue, and on the serous membranes of the abdominal and thoracic cavities, the heart, etc. The lymph glands are enlarged and injected with blood. In the pectoral form, a characteristic thickening of the interlobular connective tissue is present which tends to make the lungs appear similar to the condition found in pleuropneumonia. In the intestinal form, there is frequently a necrosis of the mucosa resulting in hemorrhages into the lumen.

There are great variations in the manifestations of both symptoms and lesions of this disease and a positive diagnosis is frequently difficult. The microscopical examination in some instances may reveal the causative agent in the blood or tissues, but this may also fail, and in such cases the only way to make a positive diagnosis is by test inoculations.

The disease being fatal in its character, attention must be directed towards preventing and checking all outbreaks. For this purpose, vaccination has been employed with uniformly good results.

The first time vaccination against hemorrhagic septicemia was resorted to, in the United States, was in an outbreak of the disease, which occurred among the buffalo in Yellowstone National Park. The method of preparation of the vaccine used in that instance has been described by Mohler and Eichhorn in an article on "Vaccination against Hemorrhagic Septicemia" in 1911. Since that time, this method has been employed on millions of animals throughout the United States, and the results have justified the use of the vaccine for preventive purposes.

In a publication on "Hemorrhagic Septicemia" by the Bureau of Animal Industry, it is stated that cattle, sheep, swine, rabbits and fowls if treated with heated cultures of hemorrhagic septicemia germs obtained from animals of the same species as that to which they themselves belong will almost invariably become protected against injections of living cul-

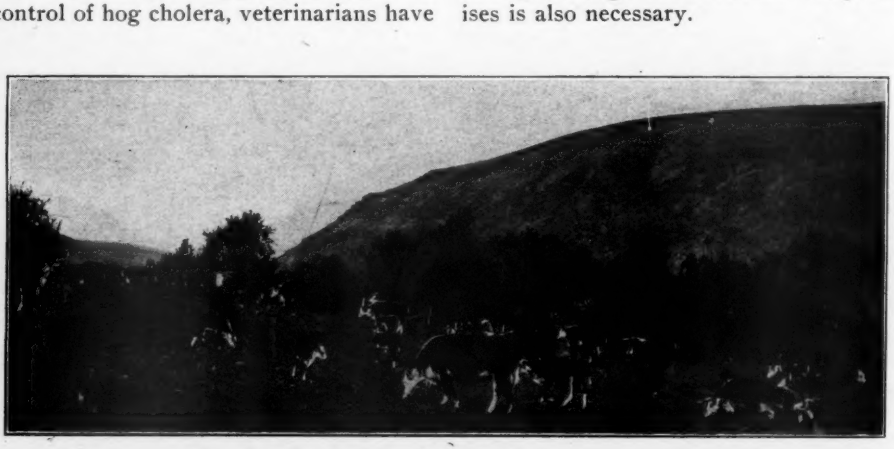
tures of the same germ, even though applied in comparatively large quantities.

In recent years, Dr. Gochenour of the Bureau of Animal Industry developed a hemorrhagic septicemia aggressin, which, upon experimental work, has proved effective in protecting cattle against fatal injections of highly virulent hemorrhagic septicemia organisms. For the purpose of aggressin production, Gochenour employed a culture isolated from buffalo, which proved very pathogenic for cattle, horses and other domestic animals. Animals infected with this culture develop very extensive subcutaneous edemas. The fluid in this edematous swelling proved to be rich in aggressins. The germ-free filtrates of these edematous fluids, when injected into susceptible animals, confer upon them a solid immunity.

By injecting the hemorrhagic septicemia cultures into horses in increasing quantities, it is possible to produce a highly immune serum, which when injected into test animals will protect them against many thousand fatal doses of active hemorrhagic septicemia cultures. It is possible with such serum to produce a passive immunity, which, however, will last for only two to three weeks. The serum may also be used for the treatment of active cases of hemorrhagic septicemia. Good results may be obtained if the serum is administered in the early stages.

Furthermore, in connection with the control of hog cholera, veterinarians have found that frequently it is advisable to use hemorrhagic septicemia vaccine or aggressin in conjunction with the serum simultaneous treatment of cholera, especially so in localities where they have learned from experience that the serum simultaneous method failed to check the disease and that hogs continued to die with marked chronic symptoms and lesions of a lung infection with hemorrhagic septicemia. In such cases, it is assumed that the serum simultaneous method will not prevent the development of the pathological process resulting from the secondary infection with the hemorrhagic septicemia organism, which naturally accounts for the continuance of losses in such instances.

Outbreaks of hemorrhagic septicemia, especially in cattle and sheep, frequently result from stockyard or stock car infections. Thus, especially during the fall and winter, numerous outbreaks occur in which cattle that have been purchased in carload lots at some of the large stockyards; hemorrhagic septicemia developing a few days after their arrival at destination. Likewise, hogs and sheep may become similarly affected with the disease. Thus, in order to prevent the occurrence of outbreaks, it is apparent that thorough sanitation of the stockyards and the stock cars is essential. In actual outbreaks, aside from the protective vaccination, a thorough disinfection of the premises is also necessary.



Coccidiosis in Cattle

By I. E. NEWSOM, Ft. Collins, Colorado
Veterinary Pathologist, Colorado Agricultural College

IT WOULD seem that the literature on coccidiosis in cattle is already so voluminous as to require no addition from me. The only excuse therefore, for this article is the request of the editor who seemingly wanted a summary of present information put into a condensed form where it might be available without an exhaustive search of the literature.

Schultz seems to have been the first to really call attention to this disease in the United States, in a paper which he read before the Oakland meeting of the A. V. M. A. in September, 1915. He had experienced coccidiosis in the Philippines and was led to examine the feces of some cattle in Washington, suffering from red dysentery. His findings seem to have been checked by no less an authority than Theobald Smith, so that they are doubtless accurate. Schultz again called attention to the disease in 1918, since which time it has been described in British Columbia, Montana, North Dakota, Colorado, Kansas, Ontario, Pennsylvania, New York, and New Jersey. Judging from the literature, one would suppose that it was limited to the temperate climate. It probably exists in the southern portion of the country but seems not to have been recorded from there.

Seasonal Distribution

In contradistinction to the disease described in Switzerland most cases have occurred in this country during the winter months, beginning in September and October and continuing even to the following May or June. As a consequence the animals were usually not on pasture but were more commonly being fed in comparatively close quarters. Neither can it be said that the disease has been especially confined to low, swampy areas as its description from such states as North Dakota, Kansas and Colorado where the annual rainfall is apt to be less than twenty inches, would seemingly contradict this view. In fact, most of the outbreaks that have come under our personal observation have had no relation whatever to low, wet ground. Not infrequently it occurs in animals in feed lots.

Age Distribution

All authorities agree that the disease is the more prevalent by far in young animals, but usually not during the first month or two of life. Old animals on the other hand, are not entirely immune as the disease has been described in milk cows from five to seven years of age. It is however, far more fatal in the young. Probably calves from three to twelve months of age are most susceptible.

Distribution of Parasites

There is much conflicting opinion on the source of infection. Some authors contend that normal animals carry the organism and are responsible for its distribution, whereas others maintain that they have not been able to find the parasite in the feces of normal cattle. Todorovic, working in this laboratory, reported having found 20% of mature cattle and 4% of calves which were slaughtered at a Denver packing house carrying the organism. This was on the basis of a single examination. If this should be confirmed it would be only in line with the findings in chickens and rabbits where it is now generally regarded that a large percentage of carriers exist among the mature. This view would also materially affect our methods of control of the disease.

Symptoms

The most prominent and most diagnostic symptom is the presence of blood in the feces. This may be present in very small quantities or may, on the other

hand, be quite profuse. Apparently it most commonly comes from the rectum as the color is practically unchanged. Sometimes the feces are covered with mucous and are rather ill-smelling. At times the animal stops eating, the abdomen is tucked up, the coat is staring and there may be considerable tenesmus. In severe cases there is a rapid loss of flesh, although the work of Osland at this institution indicates that in feeder calves that are treated medicinally, this initial loss is made up by the end of the feeding period, so that those animals that were affected, on the average made as good gains as those that showed no evidence of the disease.

In a few instances, death may take place within 24 hours after the first symptoms are noticed, apparently from hemorrhage, as the mucous membranes become very pale. There is little or no rise in temperature. In animals that survive, bleeding may be intermittent over a period of 30 days or even more. In many instances, animals will be passing considerable quantities of blood without showing any other evidence of illness, and weights may reveal no loss. The mortality is variously estimated from 2% to 50% but in treated animals it ought not be greater than the smaller figure.

Lesions

The infection seems to be confined to the large intestines and more especially to the rectum. In chronic cases, these organs are thrown into corrugated folds, the mucous membrane is thickened, edematous and often hemorrhagic. In very acute cases there may be quantities of blood in the intestinal tract, and the body tissues will have a washed out appearance. Rarely, there will be great numbers of hemorrhages under the serous and mucous membranes.

Diagnosis

Blood in the feces is almost diagnostic in itself, but the determination can be rendered positive by a microscopic examination and the finding of the oocyst. Usually the organisms are present in such numbers as to render a microscopic diagnosis very simple. If only an occasional oocyst can be found the probability is the disease may be attributed to some other malady.

Treatment

A great variety of drugs has been administered and most workers have reported satisfactory results. This seems to indicate that after the loss of the first two or three animals the tendency is for the rest of them to recover anyway. Consequently it seemingly matters little just what drugs are administered. In our own experience we found that combinations of mineral oil, salol and bismuth when given by the mouth would not stop bleeding as quickly as a strong dose of any laxative.

Rectal injections of tannic acid solutions were usually effective in stopping the bleeding, which indicates that frequently the disease is confined to the posterior bowel.

After considerable trial we came to the routine use of a pint of castor oil with warm tannic acid enemas in the more severe cases. Apparently the best results are obtained by sweeping out the massive infection. Possibly salts or some other purgative would do as well. The addition of formalin, creolin, creosote, naphthalin and other agents of this character are of questionable value, although they are almost universally used.

In the feed lots, cutting down on the grain ration where the animals would continue to eat, seemingly had no effect on the course of the disease.

Control

Since size of dose of the infecting organism seems to be the chief factor in the severity of the disease, moving the animals to new ground is nearly always effective. Where this is not feasible, as in feed lots, then the infected animals should be isolated, although our work has seemed to indicate that even the normal animals are throwing off oocysts. In a pen of ten animals, where one or two

are bleeding, usually the organism can be demonstrated in the feces of all.

For several years past we have had difficulty with coccidiosis in the calves on our feeding experiments. The pens are usually vacant from June to September or October and thoroughly cleaned and disinfected, and covered with a heavy solution of lime, and yet we experience the disease nearly every winter. Whether the infection comes in with the calves or with the feed we have not determined. There is little reason to believe that it comes in with the water, as in this case the water is from the regular city supply. Neither has it any relation to pastures as the calves are confined in very small pens, which for the most part are covered with a cement floor.

There is little evidence to show that the type of food influences the development or course of the disease. It seems probable that we shall have to accept the situation just as has already been done by the poultrymen, and attempt to carry our cattle through the susceptible period to the point where they become immune. Consequently, sanitation ought to pay in a large measure, although some of our experience has been very discouraging in this respect, as we have cleaned the pens daily and still are not able to eliminate the infection entirely.

TICK FEVER

Tick Fever, a very serious obstacle to the cattle industry of the South, has been thoroughly understood since the investigations made by Smith and Kilborne. Their work showed that the cause of the disease was a minute parasite living within the blood cells, the intermediate development of which occurred in the cattle tick—*Margaropus annulatus.*

Tick Fever is a specific infectious disease of the blood, characterized by high fever, reddish discoloration of the urine, and emaciation, with a death loss in acute form of about 85%. Enlarged spleen, engorged liver, thick flaky bile, discolored urine are found on autopsy. Care should be taken to differentiate tick fever from anthrax, blackleg, and anaplasmosis. The disease may occur outside of the quarantined area due to illegal movement of cattle or to improper use of stock cars.

Tick Fever exacts a heavy tax from the live stock interests of the quarantined area for the reason that—since May 1, 1928, the interstate shipment of tick infested cattle has been prohibited, causing a loss of from ½ to 1½ cents a pound, representing price discrimination against quarantined pens or expense of cleaning cattle. Hides are graded lower—the loss per tick infested hide being $1.25 to $2.10. There is a shrinkage in milk production; decrease in flesh and lack of development of beef animals due to tick fever infestation and the introduction of improved breeding stock is rendered difficult. The cost of eradicating the cattle tick in the average county of the South is about $40,000.00.

In 1906, when the eradication campaign began, fifteen states were infested, extending across the southern border of the United States from Virginia to California; 985 counties were under quarantine. All counties in nine of the fifteen original states have been released from quarantine, and there remains in the other six states but 198 counties to be freed. Seventy-eight per cent of the original area has been cleaned up. Real progress is being made in most of the present quarantined area.

Ft. Worth, Texas N. F. Williams,
Chief Veterinarian, Live Stock
Sanitary Commission of Texas.

LAPAROTOMY FOR TORSION OF THE UTERUS

In the February, 1929, issue of VETERINARY MEDICINE (page 48) Dr. T. H. Ferguson advises tying the legs together and rolling the cow for the correction of torsion of the uterus. He will find it much better and easier to go in through the right flank. A little local anesthetic is all the anesthesia needed.

Forest Grove, Ore. W. B. Coon.

Anaplasmosis in Cattle

By A. H. GISH, Eldorado, Kansas

ANAPLASMOSIS is enzootic in certain localities, including this one, in the late summer and early fall months. A few cases may not develop until early winter. Many outbreaks have been reported in the southern half of the United States. Whether it has recently gained access to this country or has existed unrecognized is problematic. There is some evidence to substantiate the latter assumption, although many adhere to the former, and consider that Smith and Kilborne in their pioneer study of tick fever saw and described it, but failed to recognize it as an entity separate from tick fever. It is evident that cattle in infected herds or infected territory, in time, acquire an immunity or tolerance that protects them from severe or even appreciable loss.

The mode of transmission has not been determined. Anaplasmosis is classed as a protozoan disease and some investigators have succeeded in transmitting it with ticks, but the disease is prevalent in many places where no ticks have been found in any infected herds.

Symptoms

The onset of the disease is sudden. It is ushered in with a high fever, exhaustion, unsteady gait, loss of appetite, cessation of rumination and dry muzzle. Sometimes there is nervous excitation and the animal will fight. It is easily fatigued when moved and a painful grunt is heard. Milk cows cease to produce very suddenly. As the disease progresses, the light colored tissues become noticeably icteric. The blood is noticeably deficient in hemaglobin. The percentage of hemaglobin may be as low as 25% of normal.

For a positive diagnosis make a microscopical examination of the blood from a living animal. Make blood smears with special caution. The blood should be spread in a thin film and the slides dried quickly. Stain for five minutes with Leishman's blood stain. Do not allow the stain to dry on slide before washing or small particles of precipitated stain wil confuse the examiner. Wash the slides in distilled water and dry. Examine with oil immersion lense, and if slides have been properly prepared, the *Anaplasma marginale* should be plainly visible as small dark dots in a number of red blood cells. One, two and sometimes three of these bodies may be found in a single cell. Disintegrated blood cells as found in carcasses that have been dead for some time, dust particles and crenated corpuscles make a positive diagnosis difficult.

Postmortem Lesions

All adipose tissue throughout the body is usually very icteric (lemon color). The gall-bladder is ingorged with flocculent bile. The liver is icteric and somewhat swollen and congested. The feces are hard, often covered with mucous and sometimes blood stained. Hemorrhagic petechia are often found in various parts of the body and might confuse one. The blood is visibly deficient in red blood cells.

Treatment

Avoid excitement and exertion. Provide comfortable quarters. Avoid biting flies. Inject intravenously, 25 gr. sodium cacodylate for each hundred pounds weight. An animal weighing a thousand pounds, receives 250 gr. sodium cacodylate for the first injection. Administer liberal doses of mineral oil with several gallons of water, to which add sufficient salt to make approximately a normal salt solution. Do not drench, but use the stomach tube and pump. Enemas are sometimes necessary. Avoid superpurgation. One-half to one gallon of light mineral oil and two to five gallons

of normal salt solution have given the best results in my practice. Follow this treatment with liberal doses of iron tonics or iron and arsenic compounds as arsenoferratose.

In very severe outbreaks, the initial dose of sodium cacodylate is often insufficient. The case will improve for a few days and then relapse. It is therefore advisable in such cases to follow the first treatment with arsenoferratose, or durol. I prefer to administer such compounds orally in full doses twice daily. If it is necessary to repeat the sodium cacodylate treatment before four or five days after the initial dose, one-half of the first dose may be injected. After five days from the first injection of 25 gr. per hundred pound weight, a second injection of approximately this amount may be given with a reasonable degree of safety but it should be employed only in grave cases.

Other arsenical preparations of value in the treatment of anaplasmosis are soamin and neo-arsphenamine, but when the cost and ease of administering are considered, sodium cacodylate is the drug of choice. 100 grs. of the drug may be dissolved in 50cc. of water and injected intravenously or subcutaneously without bad results.

Preventive Treatment

Little is known relative to measures to prevent the spread of the disease. Sick animals should be segregated and protected from biting insects. All carcasses should be burned promptly. All precautions should be observed to prevent the introduction of the causative agent to other animals through needles, syringes, dehorning instruments and other surgical appliances.

The injection of sodium cacodylate in doses of 25 to 30 gr. per hundred pound weight to all animals in the herd is of value in very severe outbreaks. It will check the severity of the disease, but will not eliminate it. This treatment was used on 350 head in one herd several years ago. Monthly injections were made for four months. The cattle showed no ill effects from the treatment but the disease disappeared only when the season of the year arrived when we would expect it to do so. I do not wish to leave the impression that the treatment as outlined is a cure-all for anaplasmosis. Losses will be heavy in severe outbreaks. It is the most effective treatment we have learned.

Differential Diagnosis

Hemoglobinuria or so-called red-water of cattle, sometimes occurs in conjunction with herds affected with anaplasmosis The postmortem symptoms are very similar except the wine to coffee colored urine which is not common in anaplasmosis. Microscopical examinations indicate two distinct diseases but the treatment as outlined for anaplasmosis is very valuable in these cases.

RED WATER DISEASE OF CATTLE

THIS is an acute disease of cattle but is occasionally observed in sheep. The disease is characterized by the sudden appearance of anorexia, high temperature, cessation of milk flow, depression and voiding copious quantities of very dark red urine.

Frequently a severe bloody diarrhea occurs. The course of the disease is usually 24 to 36 hours. Death occurs in more than 90% of untreated animals.

The disease occurs in swampy poorly drained pastures and mountain meadows of the Sierra Nevada Mountains of Nevada, California and Oregon. It also occurs in the Andes Mountains in Chile. Red water disease and anthrax generally occur under the same telluric conditions. However clinical symptoms and postmortem lesions are sufficient to differentiate this disease from anthrax.

The cause is believed to be an anaerobic sporulating bacillus. Inoculation of cattle and small experimental animals with young cultures of this organism produce symptoms and lesions closely approximating the natural cases.

The postmortem lesions are very striking and not readily confused with any other cattle disease. A profusion of subserous hemorrhages occur on the serous membranes of the body cavities and body organs. A large anemic infarct six or seven inches in diameter is formed in the liver. The bladder is usually full of very dark red urine. The color of the urine is due to hemoglobin liberated by the rapid hemolysis of red blood cells.

The kidneys show an acute hemorrhagic nephritis and numerous cortical petechiae.

The spleen is not enlarged but shows many sub-serous hemorrhages.

The lungs and heart show a varying degree of bloody inhibition in addition to petechial hemorrhages.

The presence of the large liver infarct and hemoglobin-uria are sufficient to differentiate this disease from anthrax.

Drug treatment of sick animals has not been successful. A curative serum was developed at the Nevada Agricultural Experiment Station which resulted in recovery of about 65% of affected cattle even though administered after symptoms were fully developed.

Serum treatment has been largely displaced by a preventive vaccination with a chemically killed bacterin.

Bacterin has been used on about 4,000 head of cattle under actual field conditions along with unvaccinated controls and appears to protect cattle in infected districts for about six months. This period of immunity is probably sufficient in a majority of instances to protect cattle during the pasturing season.

Serum for treatment of clinical cases of red water disease and bacterin for routine immunization are now available from commercial sources.

Reno, Nevada. L. R. Vawter.
Department of Veterinary Science, Nevada Agricultural Experiment Station.

BOVINE OCULAR EPITHELIOMA

The occurrence of a malignant tumor involving the eye is common knowledge. These tumors appear to be more common in range cattle, probably because cattle under range condition are not so readily accessible to veterinary service and are therefore not relieved of these new growths as are so-called native cattle.

Although the cause of tumors is still a hidden mystery, the malignant tumors that involve the eyes of cattle are frequently a sequel of infectious keratitis. Some cases have been observed in which there was no history of an attack of conjunctivitis, but these cases are relatively rare. It is of interest to note that the frequency of these tumors, at least in some range herds, is in a direct ratio to the previous prevalence and severity of outbreaks of infectious keratitis.

Although these tumors are more prevalent in mature than in young cattle, several cases have been observed in two-year-olds and an occasional case is observed in a yearling.

Generally speaking, these tumors are malignant however the length of time that elapses from the beginning of the tumor formation until the death of the animal is quite variable.

These tumors are in the beginning small, grayish pink growths firmly adherent to the cornea. The rate of growth varies but the tumor is usually at least of the size of a hen egg within six months. The neoplasm permeates the eye-ball and adjacent structures and ultimately the side of the face and head is involved.

Treatment is of little value excepting in the early stages. When the tumor is no larger than a small pea it can usually be successfully removed with little injury to the eye. If the tumor has invaded the structures of the eye and does not involve the adjacent tissues the only successful operation is enucleation. Those cases in which the neoplastic growth has invaded the eye and surrounding tissue can not be successfully operated upon.

Kansas City, Mo. A. T. Kinsley.

Infectious Conjunctivitis—Keratitis

By A. T. KINSLEY, Kansas City, Missouri

PINK-EYE, keratitis or infectious conjunctivitis, is a relatively common ailment in cattle. This disease is of considerable economic importance because it produces a marked shrinkage in adult cattle; stunting in calves, and some times the loss of one or both eyes. This disease occurs in practically every section of the country and all breeds of cattle are susceptible. The disease is more severe in cattle that are in open range where there is no shade trees or other shade provided. Calves and other young cattle are more frequently affected than older cattle.

The rapid transmission of the disease from one animal to another indicates an infectious cause. Although a specific causative factor has not been identified, the *B. pyogenes bovis,* staphylococci and *B. bipolaris,* one or all of them, can usually be obtained from the diseased eye. The infectiousness of this disease has been manifested in many cases. The disease occurs in exposed animals within a few days. An instance has been observed in which a cow affected with pink-eye was taken to a farm on which the disease had not occurred and placed in a stall between two healthy cows with the result that both healthy cows became affected within a week. Some cattlemen have believed that certain plants during their flowering stage was the cause of pink-eye, but the occurrence of the disease in mid-winter eliminates the possibility of such a cause.

Even though infection is the cause, no doubt, there are certain predisposing factors that should not be overlooked, such as dust, irritating vapors in barns, flies and glaring sunlight.

The principal symptoms are: Closed eyes and profuse lachrymation. The discharge from the eye is water-like in the beginning but later becomes yellowish and purulent and may be streaked with blood. This discharge accumulates on the margin of the lids, gluing them together. When the eyelids are opened the eyeball and the inner surface of the lids will be intensely red and swollen. The cornea is frequently covered with white colored deposit. In severe cases, the corneal surface may be eroded or ulcerated. The ulceration may result in perforation and permit of the escape of fluid and destroy the eye. In milder cases the ulcers will heal, leaving a scar which will interfere with vision. As a rule, pink eye cases show a high temperature, diminished appetite and the animals have a haggard appearance.

Prevention

The source of infection is the diseased animal, therefore, isolation of diseased animals and quarantine of additions to the herd are important factors in the prevention of this malady.

A bacterin containing *B. pyogenes,* staphylococci and *B. bipolaris* has been used quite extensively as a preventative and, according to reports, satisfactory results have been obtained.

Treatment

In the early stages of the disease it may be possible to diminish the severity of the attack by keeping the affected animal in a barn, shed or at least in the shade, thus protecting them against the injurious effects of glaring sunshine. The application of a protoiodid of mercury ointment onto the inflamed ocular mucosa is apparently of value.

This disease is usually most destructive in range cattle where it is not possible to keep the affected animals in the shade and where individual treatment is practically impossible. Ranchers usually see to it that all affected animals are driven close to the water supply and fed as some cases are practically blind for several days. All exposed animals either range or farm, should be vaccinated.

Blackleg

By LEONARD W. GOSS, Columbus, Ohio
College of Veterinary Medicine, Ohio State University

BLACKLEG is an acute infectuous disease of cattle. On rare occasions it is seen in sheep. The horse and swine are immune to it. It occurs mostly in the western half of the United States. Many of the states in the eastern half of the United States have a few localized areas where the disease is encountered at times.

Young growing cattle that are thrifty, are more susceptible than older animals, or animals which are thin in flesh. Consequently, the disease is seen mostly in epizootic form, at the season of the year when feeding conditions are at their best. This is generally in May and June and September and October. However, the disease may occur at any time of the year.

Blackleg is caused by the *Clostrydum Chauvoei*. It is a spore forming organism which is very resistant to the ordinary killing procedure of bacteria. It is endemic in the soil of the regions where the disease occurs, from time to time. Like anthrax, once a place becomes infected it is doubtful if the organisms can be dislodged.

Blackleg is a disease of the muscular tissues, as the lesions are always found in the muscular parts of the body. The lesions are seen most frequently in the anterior or posterior quarters. The thigh and shoulder are most frequently involved. It is quite unusual to see the lesion develop from an open wound, but it does occur at times. It is quite probable that the infection takes place by way of the digestive tract, with localization in a muscle.

The multiplication of the organism results in the formation of gas. This tears the muscular tissue apart, resulting in hemorrhages within this tissue. There is profuse of exudation and transudation in the vicinity of the original port of infection. Thus, a large amount of fluid and gas are contained within the lesions, which accounts for the extensive swelling. The subcutaneous tissue is of an amber color, likewise, the intermuscular tissue. The muscles within the lesion vary greatly in their appearance. Some of them, or parts, are very pale in color, while adjoining areas are a very dark red or almost black. These are the things which characterize and give the name to the disease, "Blackleg."

The symptoms of Blackleg are the swelling, which is associated with pain in the involved muscle. This results in lameness. If the lesions are confined to the legs there will be a swinging and supporting leg lameness. The affected animal lies down most of the time and may groan considerably. The temperature will rise from one to two degrees and respiration will be accelerated. Finally, the animal becomes comatose and quite dyspneic before death.

The symptoms develop rapidly and continue to increase in severity for twenty-four to forty-eight hours after they are first noticed, terminating in death in about ninety to ninety-five per cent of the cases.

The treatment consists of the use of large doses of anti-Blackleg serum. If this is given in the early stages of the disease it will save some animals. However, on account of the acuteness of the disease, treatment is not to be depended upon. On the other hand, all young animals in infected localities should be immunized at three months of age, or earlier.

There are several immunizing agents of some varying value. Blackleg filtrate, in doses of 5cc., will produce a high degree of immunity, which is of long duration. Blackleg agressin, while experimentally it does not produce as high a degree of immunity as blackleg filtrate, seems to give as good immunity as the former, as indicated by practical use. Blackleg bacterin is a more recent product and it seems to be giving satisfactory results in the field.

Sweet Clover Disease in Cattle

By A. F. SCHALK, Fargo, North Dakota
Experiment Station Veterinarian, North Dakota Agricultural College

THE so-called "Sweet Clover Poisoning" or "Sweet Clover Disease" is one of the very newest developments in cattle practice. In fact, it is an outgrowth or product of the last decade. If the disease existed prior to that time it was only of sporadic occurrence, undiagnosed, and not recorded as a specific disease entity.

Today, it stands as a recognized and established disease and no "wide-awake" practicing veterinarian, anywhere west of the Alleghanies, can afford to close his eyes to its possible occurrence in his practice, if sweet clover is used as a forage crop for cattle in his community.

In the absence of a more specific name, the term sweet clover disease appears quite justifiable at this time, inasmuch as the clinical manifestations and pathological conditions accompanying it have so far been encountered only in cases where spoiled sweet clover has entered into the rations.

Rather extensive feeding trials at the North Dakota Experiment Station, supplemented by considerable data gathered from survey work in the field, indicate quite conclusively that this disease does not develop in cattle from eating good, clean, well preserved sweet clover hay and silage. It has always, according to our observations, been associated with animals feeding on specimens of hay and silage that were molded, or, at least, possessed some degree of spoilage or damage. However, it is further noted that not all molded, spoiled or damaged sweet clover hay and silage possess the disease producing qualities. Unfortunately, the disease factor in the forage cannot be detected by either layman or scientist. Hence, mere inspection of the nature and degree of mold or spoilage of the forage is of no particular assistance in determining whether a certain specimen does or does not possess disease producing qualities.

Sweet clover disease is primarily a disease of markedly lowered coagulability of the blood of the animal and essentially a disease of resultant hemorrhages, neither of which are preceded by visible potent symptoms and clinical manifestations until the disease has been well established.

The onset of the visible evidence of the disease is usually rather abrupt and marked by great surprise on the part of the owner. From among his apparently healthy herd he suddenly observes one or two young cattle bleeding from one or both nostrils, moving with a limp or stiff gait, or showing one or possibly more subcutaneous tumor-like swellings somewhere over the more fleshy portions of the body. The animals continue to eat with normal appetite, and show no evidences whatever of fever. Contrarily, they usually present a subnormal temperature, especially in the later stages of the disease.

Undoubtedly, the disastrous condition is often brought to light in a far more striking manner when the owner experiences extremely heavy losses from excessive hemorrhages following dehorning and castrating the young animals in the late winter and early spring. In most herds where these catastrophies occur, usually from 75 to 90% of the operated animals succumb from fatal hemorrhage. In either of the foregoing conditions there only remains a history of the animals feeding on molded or spoiled sweet clover for one to invariably make an absolute diagnosis of sweet clover disease.

Decreased coagulability titre of the blood, which may reach the proportions in which "clot" phenomena is entirely lacking, is the most constant and distinguishing factor in connection with the disease. This, however, can be determined only by making special coagulation tests from time to time, which is practically a laboratory procedure.

The age of the animals and the amount of the disease producing agent in the sweet clover largely determine the period of time

lapsing between when the cattle begin feeding upon the spoiled forage and when the blood exhibits the first evidence of decreased coagulation titre and when actual hemorrhage is initiated.

Carefully checked experiments show that young cattle up to four or five hundred pounds, feeding upon exceedingly virulent specimens of sweet clover may present some decrease in coagulation of their blood within ten or eleven days. If they continue feeding such forages, they usually manifest the first evidences of hemorrhage within eighteen to twenty-five days. Specimens of ordinary virulence require longer periods of feeding before actual bleeding occurs. It ranges from 4 to 6 weeks or possibly longer, depending upon conditions.

The hemorrhages are brought about by the whole blood passing through the walls of the circulatory organs. They may occur in practically any tissues of the body, manifesting perhaps a greater predilection for the subcutaneous areas over the more fleshy portions and the subserous tissues, preferably those of the peritoneal and plural cavities. The hemorrhages range from small areas and thin ribbon-like films to large voluminous areas. Subcutaneously they are frequently present in the form of large tumor-like formations over the quarters, back and neck regions, similar in appearance to blackleg tumors. The two conditions should not, however, be confused. The blackleg swellings are composed chiefly of gas which give off a characteristic crackling sound when palpated, whereas, the swellings from sweet clover disease may range from a soft liquid nature, through a jell-like consistency to rather firm tumor-like formations. There is absolutely no gas formation nor any crackling sound in the sweet clover disease swellings.

The subserous hemorrhages are more commonly associated with the epi- and endocardia and with the stomach and intestines, where they present themselves in the form of either petechia, ecchymoses or suffusions. The conspicuous array of hemorrhagic lesions often found in profusion in many cases may prove very tempting to those inclined to fondness or weakness for hemorrhagic septicemia. While the disease is truly hemorrhagic, it is never septicemic. A few special precautions on the part of the veterinarian in checking over some of the fundamental principles of a specific infectious disease, will invariably spare one the ignominy of a grossly mistaken diagnosis in such cases.

Aside from the classical form of the disease, there are problems of equal or perhaps greater economic significance as sequelae, resulting in fatal hemorrhage from open castration, dehorning, removal of corpora lutea, parturition and the removal of retained placentae. Therefore, it is highly advisable for veterinarians engaged in cattle practice to defer all operations that can possibly be put off, until the animals have been removed from the spoiled sweet clover at least three weeks.

If the animals have not bled beyond physiologic limits, specific treatment is highly encouraging and quite successful. Drug administration, so far investigated, has been of no material aid. Likewise, ogano-therapy products have entirely failed to correct or relieve the condition. The only reliable and efficient remedy that has been established so far is transfusion of normal bovine defibrinated blood or the normal sera from the defibrinated blood. Blood from foreign species such as horses and swine as well preserved bovine blood and sera have not proven satisfactory. Sterile normal bovine sera, if properly prepared, gives some promise, but evidently requires further investigation.

Practicing veterinarians can carry out blood transfusions quite satisfactorily in the field. Only simple equipment is necessary, consisting of two, 2-quart sterile mason jars, a small funnel, about 4 feet of rubber tubing to fit and a couple hypodermic needles from 12 to 14 gauge, and a few thicknesses of sterile cheesecloth.

Draw from one to one and one-half liters of whole blood from a normal cow. Thoroughly defibrinate or whip the freshly drawn blood until all the fibrin is formed. Strain the defibrinated blood through the cheesecloth into the remaining mason jar and

slowly introduce the strained liquid into the jugular vein by means of the funnel, tube and needle. The entire procedure should be carried out in a rigidly cleanly manner.

The status of sweet clover pasture is not absolutely established. However, this institution has, during the last three years, maintained a herd of 25 young cattle on sweet clover pasture from the middle of May until early September. The major portion of these animals were horned critters and a number of them bulls. The coagulation titre of all the animals' blood was taken approximately once a week and from time to time some of them were dehorned and castrated. During this entire period, we have never experienced any appreciable lowered coagulation titre of the blood, nor has there been any noticeable excess hemorrhage following the operations. From these data, it would appear as though the sweet clover pasture is not a factor in this disease. It should be stated, however, that reports of serious hemorrhage following minor operations on cattle on sweet clover pasture have been made by practicing veterinarians and the question as to whether sweet clover pasture ever, under any circumstances, reduces the coagulability of the blood of cattle pastured upon it may be said to be as yet not definitely settled.

LOUSE INFESTATION OF CATTLE

Three species of sucking lice, *Haematopinus eurysternus, Linognathus vituli,* and *Solenopotes capillatus,* and one species of biting lice, *Trichodectes scalaris,* are commonly found on cattle in the United States. Since the periods of incubation and pre-oviposition vary in the different species and are influenced by other factors it is difficult to fix accurately the period that should lapse, on a theoretical basis, between treatments of cattle infested with several species. Experience has demonstrated, however, that the proper interval between treatments is 14 to 16 days.

When separated from their host, mature biting lice live about 7 days and the sucking species live only about four days. Newly hatched lice can live away from their host only two or three days. Ordinarily eggs are not deposited except on the host, and cattle lice do not live and propagate on other species of domesticated animals. Most cases of infestation occur from contact with lousy animals and not from infected premises.

Lice are most active, and increase most rapidly, when the weather is cold and conditions are unsuitable for dipping. It is therefore advisable to dip infested herds during the fall to insure against losses and the increased expense of winter treatment.

The usual methods of treating cattle for lice are dipping, spraying, fumigation and hand applications of liquids, powders, greases and oils. Dipping is the most successful known method of eradicating lice. The other methods are less dependable, but are of value in holding the parasites in check until weather conditions are suitable for dipping.

The dips commonly and successfully used are arsenical, coal-tar-creosote and nicotin solutions. Any of the dips may be used in the form of a spray or they can be applied by hand. The following remedies have proved to be of value when applied by hand and the treatment repeated, if necessary, in about 16 days: Cottonseed oil and kerosene, equal parts; lard, 1 pound, and kerosene, one-half pint.

Arsenical dip is probably the most effective agent known for eradicating all kinds of cattle lice. Two dippings in arsenical dip with an interval of 14 to 16 days between applications can usually be depended upon to effect complete eradication if all animals in the herd are dipped. Any of the brands of arsenical dip which are permitted by the Government for use in dipping cattle for Southern fever ticks are suitable for use in eradicating lice.

Kansas City, Kansas Marion Imes.
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Loin Disease

By H. SCHMIDT, College Station, Texas
Texas Agricultural Experiment Station

LOIN disease has thus far been observed only in cattle and manifests itself in a sudden and complete breakdown of the organs of locomotion. It occurs in that low, flat, poorly drained area in Texas bordering on the Gulf of Mexico where at present it is known to be prevalent in about 10 counties. It extends inland for some 150 miles or about as far as the low, flat area reaches. It is not definitely known whether it also occurs in other states bordering on the Gulf of Mexico.

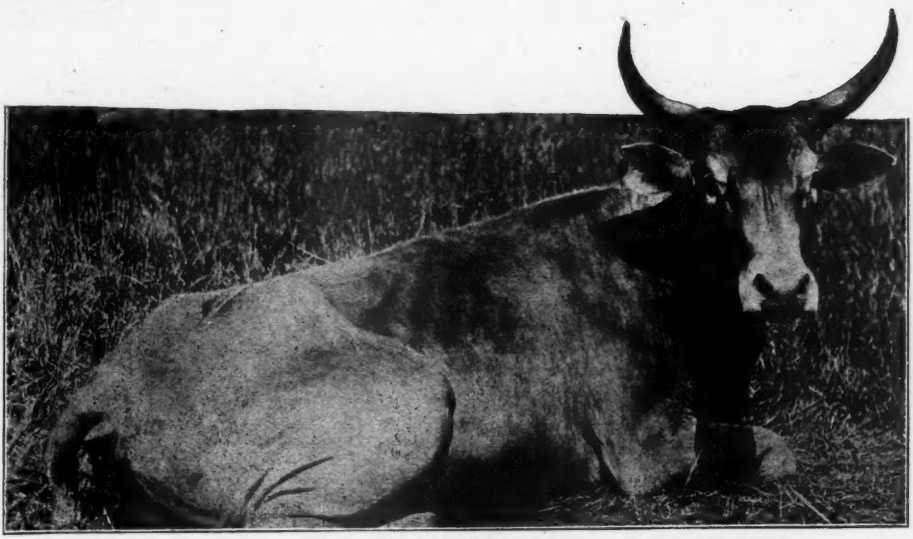
The disease occurs only in range animals over 18 months old and has never been observed to occur in animals fed a dairy ration. In an experimental herd of range animals kept under observation for five years only those animals which would chew bones contracted the disease.

BRAHMA HEREFORD COW AFFECTED WITH LOIN DISEASE
This cow got upon her feet with great difficulty, but when standing looked bright as shown in the illustration. Six hours later, she was down, unable to rise, and was killed and autopsied.

The disease has repeatedly been produced experimentally outside the loin disease area with toxic carcass material collected on the range within the loin disease area or with cultures made therefrom. It has as yet not been possible to produce the disease in any other way.

If an animal be observed in the early stages of the disease one will note that it lags behind the remainder of the herd and frequently lies down to rest. As time goes on the animal will experience increased difficulty in getting up and the time will soon arrive, usually within 10 to 12 hours, when the animal will no longer be able to rise. At first the animal will remain in a costo-sternal position, look bright and alert, body temperature and respiration normal, and will eat and drink if food and water be offered. Feces are normal at this stage but later show an admixture of considerable mucous and sometimes blood. If only a small amount of toxin has been consumed by the animal it may again be able to get up within a few hours or a few days but usually it remains down for a longer period. In this case one will observe that a paresis gradually develops in the muscles of the neck and for a short period of time, usually only for a few hours, the animal will rest its head against the side of the body much as in milk fever. Soon, however, the animal will come to lie flat on its side with outstretched legs and head, unable to rise. At this stage the animal usually refuses food and water, but a small percentage can be saved by careful nursing, recovery requiring 10 to 30 days.

Post-mortem lesions vary considerably and are quite inconstant. Thus, we may find congestion of many of the lymph glands of the body but this occurs only in about one-third of the animals. In a small percentage of the cases hemor-



Animal suffering from loin disease down only a few hours.

A cow on the coastal plains of Texas showing a typical attitude assumed when affected with phosphorous deficiency (loin disease). Practically all cases that have reached the stage of development indicated in the cut die within three days. This condition was quite prevalent in sections of the Gulf Coast region a few years ago, but is now being quite effectively prevented by providing phosphates.

rhages on the endo-cardium of the left ventricle will be found. The lungs, liver and spleen are as a rule normal. The kidneys may be congested in a small percentage of the cases, but in a much larger percentage show a distinct glomerulo-

nephritis usually without congestion. The gall bladder as a rule contains a large amount of bile and sometimes shows hemorrhages on the mucosa. Much bile and mucous is usually found in the intestinal tract. The mucosa of the fourth stomach may show congestion or small hemorrhages, although in the majority of cases it is normal. The mucosa of the small and large intestines frequently shows congestion in various degree and extent.

THE MILK FEVER POSITION IN LOIN DISEASE
Cow suffering from loin disease. This animal had been down about two days.

Since the disease is due to the consumption of toxic carcass material, the breaking of this habit is the logical thing to strive for. The basic trouble is undoubtedly a mineral deficiency of the soil and vegetation and to improve this is the desired goal. The bone chewing habit of cattle can also be broken by feeding the minerals directly to the animal and bone meal has been found to give very favorable results.

A similar disease occurs over wide areas in South Africa and has received extensive investigation by Sir Arnold Theiler. Feeding bone meal was found to be an effective preventive.

PLUMBISM—LEAD POISONING

Lead poisoning is a relatively common condition in cattle. This malady may be acute or chronic, however, there is no distinct line of demarkation between the two types of the disease.

The sources of lead as a poisoning are quite varied. Not infrequently, lead in the form of a fine powder may escape in the fumes and smoke from mines, smelters, furnaces, etc., thus contaminating vegetation. This is a frequent source of lead in the chronic type of plumbism. The usual source of lead in lead poisoning is from recently painted barns, fences, hay racks, feed boxes, buckets and other equipment used in cattle barns and yards.

Sufficient lead to produce poisoning is some times obtained by cattle that lick discarded paint kegs and buckets, or paint that is discarded as waste may be a source of lead poisoning. Lead packing from joints of pumps, engines and other machinery may cause trouble. Spray from rifle bullets on target range has been found to cause lead poisoning. Less frequently lead poisoning may occur as a result of contaminated feed or

water that is stored or passed through lead lined pigs, tanks or pipes.

Symptoms

In chronic lead poisoning there is usually a stunted growth, emaciation, some disturbance of breathing, incoordination and if such animals continue to obtain the lead, death ultimately ensues.

Acute lead poisoning is manifested by colicky pains, champing of the teeth, dripping saliva from the mouth, disturbed vision, incoordination depression, stupor and death.

The foregoing symptoms of acute lead poisoning usually occur within a few hours after the poison has been consumed. In a few cases the symptoms may not develop for some time after the lead has been consumed, because of its accumulation in the rumen in masses of feed.

Lesions

In chronic lead poisoning there will be general emaciation, rough coat, in young animals there will be under-development, and there may be a so-called blue line along the gums. In acute lead poisoning there is usually a gastritis and enteritis of varying intensity. Infrequently cases of lead poisoning require chemical analysis and in either acute or chronic form there will be little difficulty in demonstrating the presence of lead, particularly in the content of the rumen and in the liver and kidney.

Treatment

The treatment of lead poisoning entails first the prevention of animals from obtaining more of the poisoning. In the acute cases, medicinal treatment is frequently of no avail, as sufficient of the poison has been absorbed to cause death before drugs can produce a counter action.

Saline purges such as Epsom or Glauber's salts with diluted sulphuric acid, milk and eggs, are indicated.

In addition, stimulants will probably be of value. In the chronic cases potasium iodid will be found of some value.

Kansas City, Mo. A. T. Kinsley.

BRISKET DISEASE

Brisket disease is a malady that occurs in cattle that are kept on high mountain ranges. It was reported by veterinary authorities in Colorado some years ago and more recently in New Mexico the Animal Industry Department has reported the existence of this disease in their state. Many efforts have been made to relieve the affected cattle by moving them to a lower altitude, but thus far the change of altitude has not produced a cure in the animals affected.

Of course the condition concerns relatively few veterinarians but it is of general interest because of its peculiar cause. Animals rarely suffer from heart involvement except as a result of infection or traumatisms. Here however is an ailment of the heart not related to either infection or traumatism.

This is not an infectious or contagious disease, according to Glover and Newsom of the Colorado Experiment Station, and there is no danger of the transmission of the disease from an animal affected to other in contact animals. The condition is apparently caused by high altitude. A number of cattle have died as a result of this disease in the higher regions of the Rocky mountain section. In one herd, 60 of 99 animals died of the disease.

Brisket disease is manifested by difficult breathing, gradual loss of condition and cough. In some cases in the later stages, there will be found a swelling in the brisket region that is of a dropsical character.

The relief of the disease is not difficult if the cases are obtained in the early stages. They are successfully treated by the use of heart stimulants. In the range sections of the mountainous areas cases usually are not observed until they are far advanced and seldom can be relieved.

Some of the investigators have recommended that cattle that are ranged in the high altitudes should be bred in the relatively high altitudes. That is, cattle should not be taken from low altitudes to extremely high altitudes.

Kansas City, Mo. A. T. Kinsley.

Shinnery Poisoning—Scrub Oak Poisoning

By A. T. KINSLEY, Kansas City, Missouri

DURING the last twenty years, there have been several reports of the loss of cattle that had eaten scrub oak (shinnery) leaves.

Shinnery poisoning was observed in a shipment of some 4,000 three-year-old steers that had had access to scrub oak in Texas in the early spring, and later shipped to the flint hill district in Southeastern Kansas for summer pasture. Two or three days after arrival several steers were found dead and within a week over one hundred died and there were several others seriously affected, many of which died later.

Several reports were received during the latter part of April and first part of May, 1925, of shinnery poisoning in cattle that were shipped from so-called shinnery areas in Oklahoma and Texas to Kansas pastures. One outbreak was investigated in which about 600 head of steers were shipped from Oklahoma to South central Kansas, in which 30 died of shinnery poisoning within the first few days after arrival and about 35 more were noticeably affected.

Cause of Shinnery Poisoning

There are several hundred varieties of oak in the United States, however, it is probable that only a few varieties are poisonous. According to reported investigations the leaves of certain scrub oak are poisonous at least at certain times of growth. Poisoning has been attributed to at least two species of oak, one of which occurs in Utah and the other in New Mexico, Texas and Oklahoma. The species occurring in New Mexico, Texas and probably in Oklahoma, is commonly called shinnery. It is generally conceded that the most severe poisoning occurs in early spring and is attributed to eating the buds and young leaves. Poisoning usually occurs in pastures in which there is scarcity of grass and the oak buds are available several days before the grass.

Symptoms of Shinnery Poisoning

The symptoms of this condition though fairly constant may be somewhat variable, probably due to the condition and disposition of the animals. Usually, the first symptom is constipation. Small hard balls of fecal matter are passed at infrequent intervals. As the disease progresses and usually within two or three days after the first symptoms have been observed the fecal balls are covered with mucus and blood. In some cases quantities of a stringy blood tinged mucus is passed at infrequent intervals. If the animal survives for four or five days after the onset there may be a diarrhea. The affected animal is gaunt, has a rough coat and the muzzle is dry and may be cracked. They extend the head, the eyes are sunken and they show marked depression and discomfort. They will not eat but have an increased desire for water. It has been found that practically all shinnery animals will remain around the water holes because of their increased thirst. The temperature is practically normal but the heart action is apparently depressed. The breathing may be slightly faster than normal but there is no nasal discharge or cough. The autopsy findings of shinnery poisoning consist of congestion, erosions and hemorrhages from the lining membrane of the true stomach and intestines.

This condition may be mistaken for hemorrhagic septicemia and arsenical poisoning. A history of the cattle having had access to scrub oak and a laboratory examination, will suffice for positive diagnosis.

Treatment has proved unsuccessful.

Arsenical Poisoning

By A. T. KINSLEY, Kansas City, Missouri

POISONING of cattle by arsenic, although not of common occurrence, is of considerable interest and of some economic importance. Many cases of rather extensive losses of cattle that have been dipped in arsenical solution in which there was excessive arsenic, have been reported. One notable instance is of record in which several hundred head of cattle that had been exposed to tick infestation were dipped in an arsenical solution in which the quantity of arsenic was doubled. After dipping, these cattle were shipped approximately 300 miles. At time of arrival at destination, several were dead in the cars, others showed extreme depression, stupor and many others that were apparently normal after consuming quantities of water died. The total loss of this shipment of cattle was some 200 head.

Other instances have been recorded in which rather extensive losses have occurred in cattle that were permitted to graze in orchards that had recently been sprayed with arsenical solutions. Occasionally, losses are reported in cattle that accidentally gain access to railroad right-of-ways on which the railroad companies have used a preparation of arsenic to destroy the weeds. Less commonly, arsenical poisoning occurs as a result of the accidental eating of rat poison containing arsenic or due to the ingestion of water contaminated with arsenic from chemical plants.

Several years ago, there were extensive losses of not only cattle, but of other domestic animals in Montana; primarily, according to records, due to arsenic deposits on vegetation from the smoke of smelters, particularly in the copper mining districts.

Symptoms

The symptoms of arsenical poisoning vary according to the quantity of arsenic consumed and whether or not it is ingested or applied to the skin in a form of a spray or dip. The usual symptoms manifested in cattle that have been dipped in vats containing excessive quantities of arsenic consist of colicky pain, diarrhea, marked depression and dermatitis. These symptoms come on quite rapidly and fatality depends upon the amount of arsenic actually absorbed through the skin. Ingestion of arsenical poisoning has a similar chain of symptoms excepting in the acute form, skin lesions are rarely present.

Postmortem Findings

The lesions evidenced in acute poisoning due to arsenic consists of acute gastro-enteritis, usually glomerular nephritis and frequently dermatitis.

Chronic arsenical poisoning is manifested by unthriftiness more or less articular involvement, subacute or chronic gastro-enteritis and in some cases there is a chronic nephritis. The carcasses of animals that have died of acute arsenical poisoning are apparently quite well protected against putrefaction, particularly will it be noted that there is little gas accumulation in the digestive tube in an animal that has died of acute arsenical poisoning.

Treatment

Treatment of acute cases of arsenical poisoning is not satisfactory unless the case is treated within a very short time after the arsenic has been consumed. In those cases that have consumed quantities of arsenic the usual remedy is the precipitated tincture of iron chlorate with sodium carbonate. This should be followed by the use of demulcents which, to some extent, will relieve the acute irritation in the digestive tract.

Cornstalk Disease

By A. T. KINSLEY, Kansas City, Missouri

THERE are reports of the loss of cattle in stalk-fields each year throughout the corn belt. These losses have been more or less regular for the last fifty years. Some have designated the condition as cornstalk disease, while others contend it is not a disease but merely digestive derangement or poisoning of cattle.

Occurrence

Cornstalk disease is peculiar in many ways. It may occur in the same area for two or three successive years and suddenly disappear and is almost certain to recur again at a later date. Instances have been recorded in which the majority of the herd of cattle died before they could be driven from the cornstalk fields. The disease most frequently occurs in cattle within a few hours or at most within one or two days after the animals have been placed in a stalk-field. Losses rarely occur in cattle after they have been in the field for a week or longer. Several instances are recorded in which a given herd of cattle have foraged in a particular stalk-field for a month or six weeks without any losses and immediately after turning this same herd of cattle into an adjacent stalk-field losses occurred. The disease occurs equally frequent on bottom and uplands and its occurrence has no relation to the nature of the soil.

Cause and Nature of Disease

The exact cause and nature of this malady has not been determined. It would appear that the name "cornstalk disease" probably includes more than one actual disease condition. By carefully analyzing the history, symptoms and postmortem findings of cattle that have died in stalk-fields it is possible to identify three different conditions although the identification may not be complete.

One condition may be designated as a food poisoning. In this condition the affected animal dies, in some instances, within 15 or 20 minutes after entering the field. They evidence marked distress and apparently die of suffocation due to respiratory arrest. The cause of this sudden death is probably some poison that occurs in some portion of the fodder or the poison may be formed in the animal body by the action of ferments. Death is inevitable in these cases and invariably occurs within a few minutes after the first symptoms occur.

A second type of disease that occurs in cattle in stalk-fields is due to mechanical interference or obstruction in the digestive organs, due to the accumulation of quantities of relatively indigestible materials. This produces digestive derangements which may or may not be fatal, depending upon whether or not it can be removed. Cattle affected with this form of the disease are sluggish and have little, if any appetite. They may lie around in the yard for two or three days and ultimately recover. If any portion of the digestive tube is occluded by impacted husks, leaves or stalks and the obstruction persists, the animal will die. Some of these cases may be relieved by operation and the removal of the impaction. This operation is not ordinarily successful unless it is done in the early stages of the disease.

A third form of cornstalk disease is, according to the findings in various sections of the country, due to infection. The infecting agent in these cases has been identified as a member of the hemorrhagic septicemia group. This is probably the most common form of so-called cornstalk disease. Cornstalk hemorrhagic septicemia is usually acute and is rapidly fatal. No doubt, many of the cases of cornstalk disease began as digestive disturbances which diminishes the resistance of the affected animal and the ever present hemorrhagic septicemia organism finding conditions favorable develops

rapidly and produces death in a short time.

Prevention

As previously stated, cornstalk disease probably includes at least three distinct conditions, namely,—acute poisoning, impaction and hemorrhagic septicemia.

The only known method of preventing the acute poisoning condition is to keep cattle out of the stalk-fields. The feed value of the fodder in stalk-fields can not be wasted and therefore it has been suggested that in the beginning cattle should be fed prior to being turned into stalk-fields, and they should be permitted to remain in the field for only a short time, not to exceed one hour at first and thus gradually become accustomed to this feed. This practice should be continued daily for ten days to two weeks. The report of those that have tried this plan seems to indicate that the losses from this particular cause has been materially reduced.

To prevent the second form of cornstalk disease, that is impaction, it is necessary to cause the cattle to consume extra quantities of water. This may be accomplished by supplying small quantities of grain feed with which there is admixed relatively large quantities of salt at night, thus increasing the desire for water of which ample quantities should be available.

The hemorrhagic septicemia form of cornstalk disease can be prevented by having cattle immunized against that disease. The length of the immunity produced by the present methods are relatively short and should losses occur after one month or longer after vaccination the process should be repeated. It should be clearly understood that immunization against hemorrhagic septicemia will not prevent the losses due to poisoning or impaction.

CATTLE GRUBS OR OX WARBLES

Cattle grubs, *Hypoderma bovis,* found only in the Northern States, and *H. Lineatum,* occurring in every State in the union cause annual losses estimated at fifty million dollars. The U. S. Bureaus of Animal Industry and Entomology are conducting field experiments and life-history studies with the object in view of finding some satisfactory method of control and eradication.

Incomplete returns show that there were removed from 48 Colorado herds, totaling 3,616 head of cattle, 122,283 grubs, or an average of about 34 grubs per animal. The largest number of grubs found in any one animal was 364. The average labor cost of removing and destroying the grubs was about 17 cents per head and one man could handle about 3 head of cattle per hour.

The method of eradication used in most cases was to remove the grubs with small alligator forceps and destroy them. To lessen the danger of spreading infection from one lesion to another it was found necessary to disinfect the forceps each time they were used in a grub opening. This was accomplished by providing a wide-mouth bottle partly filled with denatured alcohol, and as the grubs were extracted they were deposited in the bottle and the jaws of the forceps were immersed in the alcohol in one motion.

Experiments are being conducted with vacuum extraction apparatus, effects of certain insecticides on the grubs, and the killing power of oils on the eggs.

Kansas City, Kansas Marion Imes.

Self Sucking Cows

By S. L. STEWART, Olathe, Kansas

A SELF sucking cow is a constant source of annoyance and a definite liability to the owner, unless in some way she is prevented from daily indulging her vice. There are a number of devices which have been contrived to prevent cows from sucking themselves, but all are more or less a nuisance and a failure. One of the most unsightly is the 2x4 scantling tied to the head stall of the halter and the other end to a surcingle around the body; another is a piece of a slat hog fence fastened around the cow's neck, to prevent more than slight lateral flection of the neck.

A veterinarian should always offer his services when he sees such cases and render a client a valuable service by operating on the cow.

There are at least three operations that usually are successful. 1. Removal of half of the free portion of the tongue; 2. Removing a V-shaped piece from the free portion of the tongue; 3. A horizontal incision through the free part of the tongue.

Field Operations

No. 1. A tongue operation of any importance is very painful and the cow should be anesthetized, preferably with chloral hydrate, giving from 2 to 4 ounces in a quart of warm water. The amount given depends upon the cow, size, condition and whether she has been gaunted before the time of operation. Even then some cows will respond to the action of chloral much more than others, chloroform may be used to finish anesthesia if needed.

When the cow is ready she should be firmly secured by casting on the right side, tying all the legs, securing the head with a good bull lead, the attendant holding the bull lead and the left ear, to immobilize the head. The operator should take a position in front of the cow's head, then with the left hand grasp the tougue, pulling it well out of the mouth, then with a large pair of serrated scissors, split the tongue in the median line back to the frenum. Then with a transverse incision in front of the frenum linguae, cut off the left half, ligate the lingual artery and release the cow.

Some prefer to cauterize the cut surface of the tongue, after the piece has been removed. Others use a knife instead of scissors to operate, and first cut and turn back the mucous membrane from the line of incision, and when the piece of tongue has been removed, the mucous membrane is drawn down over the cut edge of the tongue and sutured. This makes a fine looking, finished operation, but the mucous membrane is not very strong and the movements of the tongue will soon cut the stitches through and release the membrane and if the operator will look at the cow's tongue the second or third day afterward, the tongue will look ragged and frequently heals with a roll of scar tissue which sometimes causes a stay for the tongue sufficient to allow the cow to again suck herself.

No. 2. The V operation is performed the same as before described, except that a V-shaped piece of the tongue is removed, the apex of the V extending well back to the frenum linguae, removing the median raphe.

No. 3. The horizontal operation consists of passing a long bladed bistoury in the border of the tongue in front of the frenum linguae, to the opposite side, then split the muscles from that point forward to the apex, at all times being careful not to cut the mucous membrane anywhere, except at the place where the bistoury was introduced into the tongue, somewhat as tendonotomy on horses is performed.